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CARCINOMA OF THE LUNG: BRONCHOSCOPIC ASPECTS*

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BRONCHOSCOPY is the most important method of diagnosis in primary carcinoma of the lung. Such new growths appear to be on the increase and now cause from 8 to 18 per cent of all deaths due to cancer, as reported by Jaffe,[1] Koletsky,[2] Simons,[3] Graham,[4] and Ochsner and DeBakey.[5] Approximately 75 per cent of such tumors arise in the major bronchi, as shown by Tuttle and Womack,[6] Gebauer,[7] Broyles and Fisher,[8] and Overholt and Rumel.[9] Lesions so situated can be visualized and biopsied bronchoscopically.

In 1933, before the advent of pneumonectomy for cancer of the lung, bronchoscopic investigation of such cases was infrequent. The high percentage of positive diagnoses that would be obtained by this method of investigation if all persons suspected of having pulmonary neoplasms were studied endoscopically is still not generally appreciated. In 1939, Taylor[10] stated that "confirmation of lung tumors is not usually available preoperatively. . . ." This statement may have been true, but need not be true today if all patients with unexplained pulmonary symptoms are studied bronchoscopically.

The patient with a change in bowel function rightfully deserves a proctoscopic examination. Abnormal urinary symptoms often indicate the desirability of cystoscopic studies. Likewise, the patient with unexplained pulmonary symptoms cannot be considered fully investigated without a bronchoscopic inspection of the trachea and bronchial tree.

Establishing a verified diagnosis of cancer at an early stage of the pathologic process should not be so difficult in neoplastic lesions of the lung as in other internal organs. In a personal series of 62 cases with histologically verified primary neoplasms of the lung, the lesion was seen and a positive preoperative diagnosis of cancer made from the biopsy specimen in 46 cases (74 per cent). In contrast with this, cancers of the kidney, gall bladder, pancreas, stomach, brain or colon, with the exception of the rectum and sigmoid, are practically never histologically verified before exploration. The high percentage of preoperative positive biopsies in cases with a pulmonary neoplasm should make this lesion one of the most favorable types. This report is presented to demonstrate the value of bronchoscopy in the diagnosis of pulmonary tumors and its application to the determination of operability and expedition of therapy.

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PRESENT-DAY BRONCHOSCOPIC EXAMINATION

Endoscopic investigation of the tracheobronchial tree should not be delayed because of hesitancy on the part of the physician or fear on the part of the patient. It is not an ordeal to be endured, but rather it is an examination that can be performed easily under local anesthesia, with minimal discomfort and with little inconvenience to the patient. Therapeutic bronchoscopies may be satisfactorily managed in an outpatient department, but diagnostic investigations are preferably done as a hospital procedure. The patient is admitted the day before and is ready for discharge the day following the examination. Such a routine permits adequate investigation and preoperative medication, the latter being a valuable adjunct to local anesthesia. Patients are taken to the operating room conscious, but relieved of the sense of anxiety and fear by a barbiturate two hours previously, and by morphine and scopolamine subcutaneously one hour preoperatively. Topical application of cocaine or Larocaine to the hypopharynx and each pyriform sinus is supplemented by the intratracheal instillation of a few cubic centimeters of a diluted solution of the same agent. Painful impulses from the upper respiratory tract are thus abolished, the irritative sensation of a foreign body in the tracheobronchial tree is alleviated, and the cough reflex is suppressed.

Proper teamwork is essential for this type of

endoscopic work. Inadequate preparation, inexperience and lack of teamwork on the part of the operator and assistants are responsible for the common misconception that bronchoscopic examination is a severe ordeal for the patient. With the patient in the dorsorecumbent position, with the head and shoulders projecting over the end of the table, the head is held forward and extended on the cervical vertebrae. In this position, a straight line is formed from the open mouth to the trachea. A straight, nonflexible tube, such as the bronchoscope, can then be inserted into the trachea without trauma.

A careful and complete examination of the trachea and the visible portion of the bronchi can be carried out in a few minutes. A definite routine of inspection, including examination of all major bronchi, should be followed to obtain all the information possible. Patients frequently express surprise at the lack of discomfort, and in many cases they do not even remember the procedure the following day, especially if scopolamine has been administered preoperatively. When the investigation is performed carefully, it is practically without risk.

Indications for Bronchoscopy

Bronchoscopic examination is indicated in every patient who is suspected of having a primary pulmonary cancer, except for the patient with obvious widespread metastases. Practically all patients with neoplastic lesions have some respiratory symptom. Ninety-eight per cent of the series reported by Overholt and Rumel[9] had definite symptoms that indicated an abnormality of the respiratory system. They found cough to be the most frequent symptom (87 per cent); fever was next (53 per cent); and chest pain or discomfort was third (44 per cent). Hemoptysis, marked weight loss and symptoms due to metastatic lesions are usually late signs and should not be awaited before institution of adequate investigation. The disease is most frequently encountered in the fourth and fifth decades, but the extremes are not uncommon. The age of the patients in the present series varied from eighteen to sixty-seven years.

It is not the intention of this report to promote the idea that all patients with pulmonary symptoms should have a bronchoscopic examination. However, all patients with unexplained pulmonary symptoms do warrant this investigation. Cases of pneumonia that are atypical and do not respond to the usual therapy in a few weeks may be due to bronchial obstruction by a tumor. Recurrent pneumonia of the same lobe may be due to intermittent atelectasis of that lobe. Development of a respiratory wheeze, especially in a patient without past allergic history or in one who does not respond promptly to allergic therapy, necessarily places that patient in the class in which cancer is suspected.

The etiologic factor in most pulmonary lesions becomes apparent after a careful history has been taken, after a complete physical examination has been made, and after adequate roentgenologic studies have been secured. Sputum, if obtainable, should always be examined for both acid-fast bacilli and other organisms.

It is the patient who is still not diagnosed after performance of the above studies who warrants bronchoscopic examination. Even a roentgenologic diagnosis of pulmonary cancer is not sufficient, since a verified diagnosis cannot be made by this method. Too often, the physician becomes discouraged and goes no farther after receiving a report of primary lung cancer from the roentgenologist. Furthermore, the extent or operability of a given lesion may be misleading by roentgenography. Bronchoscopy is the most accurate way of determining both these factors.

Classification

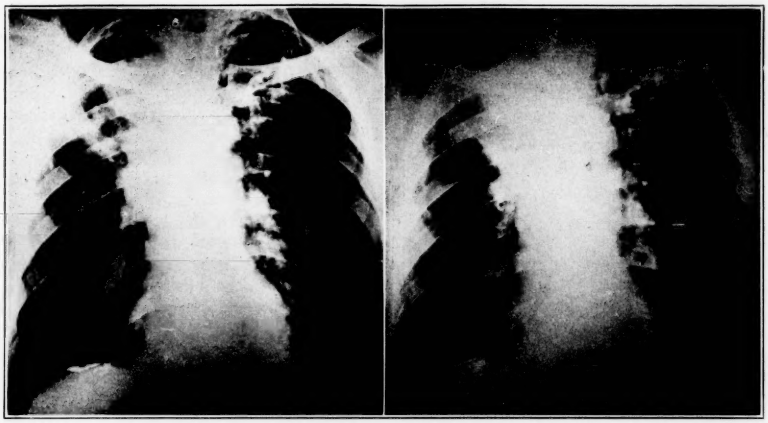
From the bronchoscopic viewpoint, pulmonary carcinomas may be conveniently divided into two groups, central and peripheral, depending on the site of origin of the lesion. The central or stem-bronchus group (Figs. 1 and 2) includes those arising in the larger air passages, that is, proximal to the bifurcation of the bronchi of the third order. In other words, these lesions arise in the main bronchus, in the lobar bronchi or in the proximal portion of one of the lobar bronchial subdivisions. Lesions in this central group comprise approximately 75 per cent of all primary neoplasms. If the tumor itself can be seen endoscopically, it necessarily falls in the central classification. Peripheral tumors include all those arising beyond the proximal segment of a bronchus of the third order. They cannot be directly visualized by bronchoscopy, although frequently a deformity of the bronchus may be noted owing to an inward displacement by pressure of the tumor mass. Such an observation is strong presumptive evidence of neoplasm, although tissue may not be obtained for histologic examination. Bronchoscopy is the most accurate way of determining whether the lesion is central or peripheral.

For three reasons, tumors falling in the central group of neoplasms seem more favorable from a therapeutic standpoint. First, a new growth in a major air passage produces symptoms earlier than

one distally situated, since it soon encroaches on the lumen of the bronchus and thus gives rise to symptoms; peripheral tumors usually have to be of considerable size before structures are involved that will cause the patient to realize there is something abnormal. Secondly, lesions arising in main or lobar bronchi can be visualized and though there was no roentgenographic evidence of disease.

In Late Primary Lesions

As soon as the growth reduces the lumen of the bronchus appreciably, abnormal physical signs and roentgenographic abnormalities appear. From



A B

FIGURE 1. *Roentgenograms of Chest.*

A *shows partial atelectasis of the right upper lobe. Owing to the presence of diffuse bilateral calcified foci, the patient was treated for pulmonary tuberculosis in a sanatorium for seven months, in spite of a negative sputum.* B, *taken six months later, shows progression of the lesion to complete atelectasis. A diagnosis of epidermoid carcinoma was established by bronchoscopic biopsy.*

biopsied bronchoscopically, thus permitting an earlier verified diagnosis, with resultant improvement in treatment. Thirdly, there is evidence (Tuttle and Womack[6]) to show that tumors in the central location grow more slowly and metastasize later than lesions situated peripherally.

USES OF BRONCHOSCOPY

In Early Primary Lesions

A small submucous bronchial lesion may not produce pulmonary signs or symptoms. When such a tumor extends intrabronchially or breaks through the mucosa, it produces cough or, if ulceration is present, hemoptysis. Depending on the size and configuration of the neoplasm, roentgenographic findings may or may not be present. Few cases are observed at such an early period, yet the lesion may be visible bronchoscopically. In one case in this series, investigated because of hemoptysis, a diagnosis of pulmonary carcinoma was established by bronchoscopic biopsy even meager physical and roentgenographic findings to most striking signs may represent a very short period in the tumor cycle. When a major bronchus becomes occluded, that portion of pulmonary parenchyma supplied by it becomes atelectatic; hence, marked signs become apparent. The extent of the findings on physical and roentgenographic examinations is not a criterion of the extent of the disease, since a mass 2 or 3 cm. in diameter located in a main bronchus may produce atelectasis of the entire lung. If such a tumor were primarily intrabronchial, it might well not have extended beyond the realm of resectability. Bronchoscopic visualization of the tumor gives a much more accurate knowledge of its extent than either physical examination or roentgenography.

In Metastatic Lesions

The lungs are frequently the site of metastatic implants from distant organs. As a rule, pulmonary metastases reveal themselves as multiple, well-defined, discrete shadows in the pulmonary

parenchyma and cannot be visualized bronchoscopically. On occasion, an implant may involve the wall of a major bronchus and produce roentgenographic findings of atelectasis, which cannot be differentiated from those produced by a primary bronchiogenic neoplasm.

In two cases, I have obtained positive biopsies bronchoscopically in tumors that were not bronchiogenic in origin. One was a lymphoma with involvement of the left lower-lobe bronchus, and the second was a metastatic implant from a mel-

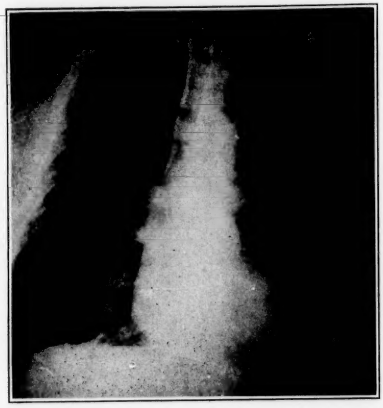


FIGURE 2. *Roentgenogram of Chest.*

In this patient, who was sixty-seven years of age, with a history of a moderately productive cough of two months' duration, a presumptive diagnosis of bronchiectasis had been made. Bronchoscopic examination revealed a primary bronchiogenic carcinoma. Note the haziness of the right lower-lung field. Pneumonectomy was successfully performed.

anotic sarcoma of the eyelid, the original tumor having been removed four years previously.

Other Uses

Bronchoscopy should not be limited to the one function of establishing a histologically verified diagnosis. Information of great value in determining the plan of treatment can be obtained by a complete bronchoscopic visualization of the trachea and major bronchi.

Determination of operability. Radium, roentgen rays and surgery are the three methods available for treating primary carcinoma of the lung. Reports have appeared in the literature by Leucutia,[11] Brock[12] and Leddy and Moersch[13] of five-year cures by radium or roentgen-ray therapy, or both. Surgery, by far, gives the greatest hope for cure.[4, 9, 14] Hence, it is necessary to have indisputable evidence that the lesion cannot be removed surgically before denying the patient this chance for surgical cure. Metastases to bone, brain and other organs frequently take place. If such extrathoracic extension cannot be demonstrated, evidence of local extrapulmonary involvement should be searched for. One cannot always be certain bronchoscopically that a given lesion is operable. Various findings, however, may demonstrate that extrapulmonary extension has taken place, and that the lesion is therefore inoperable. Bronchoscopy may thus save the patient a thoracic exploration.

Bronchoscopic evidence of inoperability may be grouped under two headings: local intrabronchial direct extension to an unresectable area of the tracheobronchial tree, and extensive paratracheal lymph-node involvement.

Lesions originating in either the main-stem or the right upper-lobe bronchus may extend so as to invade the wall of the nearby trachea. Such

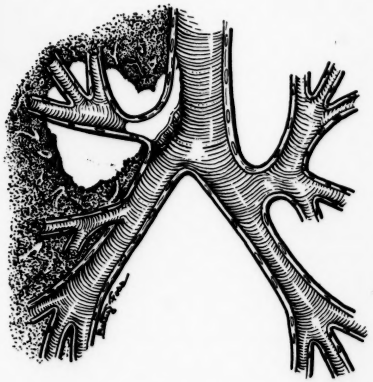


FIGURE 3.

This drawing illustrates how a lesion originating in the vicinity of the upper-lobe orifice may, by direct extension, invade the nearby trachea and become unresectable.

an extension can be visualized bronchoscopically and inoperability determined (Fig. 3). A not infrequent finding is involvement of the contralateral main-stem bronchus. This appears to take place by extension from the infracarinal lymph nodes (Fig. 4).

The lymphatic drainage of the lung is primarily

centripetal. When an extrapulmonary lymphatic extension takes place, the paratracheal lymph nodes are usually the first to be involved. Such involvement, if extensive, causes the angle between the two main-stem bronchi to become more obtuse.

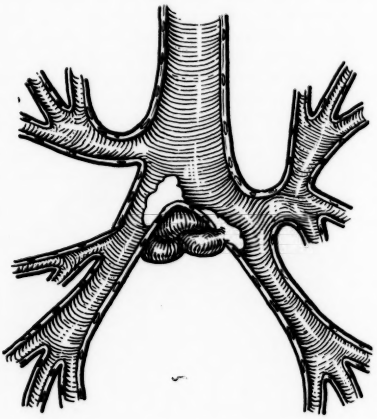


FIGURE 4.

This drawing illustrates extension to the contralateral bronchus by direct extension from an infracarinal lymph node.

Fixation may be present, just as a so-called "frozen pelvis" is found in advanced cases of carcinoma of the cervix. The extent of mediastinal fixation can be determined by palpating these structures with the tip of the bronchoscope. The finding of a widened carina with hardened and fixed structures, as determined bronchoscopically, is prima facie evidence of mediastinal metastases, especially if a positive biopsy from the primary site has been obtained (Fig. 5).

Therapeutic value of bronchoscopy. The usefulness of bronchoscopy is not confined to diagnosis or determination of proper treatment. It may be of therapeutic value itself. Frequently, the bronchial obstruction produced by a neoplasm causes an infectious process in the segment of lung supplied by that bronchus. Gentle bronchoscopic dilatation or even removal of a portion of the tumor mass provides better drainage and may give relief from the septic process. Repeated bronchoscopic aspirations are occasionally indicated in such cases to improve the patient's general condition preoperatively. In certain cases not amenable to surgery, radium or radon may be used in an intrabronchial applicator introduced bronchoscopically. This procedure has been recommended as a means of destroying the intrabronchial portion of the tumor to provide drainage for the outlying pulmonary parenchyma. My experience with this type of treatment is too meager to permit drawing conclusions.

BRONCHOSCOPIC RESULTS

Of 62 patients* with histologically verified pulmonary tumors examined bronchoscopically, 49 were men and 13 women, a ratio of 4:1. Their ages ranged from eighteen to sixty-seven years, with an average of fifty-two years.

The tumor originated in one of the major bronchi in 46 patients, and was more peripherally located in 16. In 46 patients (74 per cent) — 45 (98 per cent) with central tumors and 1 (6 per cent) with a peripheral tumor — a biopsy and subsequent histologic diagnosis of neoplasm were obtained. The patient with a central tumor in whom a biopsy was not obtained had an inoperable lesion originating in the left upper-lobe bronchus (determined at autopsy). At the time of bronchoscopy, there was so much fixation of the mediastinal structures that the bronchoscope could not be introduced as far as the upper-lobe orifice; therefore, tissue was not obtained by bronchoscopy in this particular case, although a clinical diagnosis of inoperable bronchiogenic carcinoma was established.

Location of Tumor

The frequency of involvement of each bronchus in the series of 46 central tumors was as follows: right upper, 10; right main-stem, 10; right lower, 9; left lower, 7; left main-stem, 5; left upper, 3; and right middle, 2.

Thirty-one, or 67 per cent, originated on the right side, and 15, or 33 per cent, on the left. Inasmuch as satisfactory closure of the bronchus is technically the most difficult part of a pulmonary resection, it is unfortunate that the region of the right upper-lobe bronchus is most frequently involved. This bronchus arises from the right main-stem bronchus, from 1.5 to 2.0 cm. from the trachea. A growth originating in this area may early invade the wall of the neighboring trachea and thereby become technically unresectable (Fig. 3).

Malignant tumors should be removed with a margin of uninvolved tissue and all available lymphatics, since extension is most frequently by this route. The treatment of pulmonary cancer is comparable to that of cancer of the breast. Axil-

*Some of these patients have been previously reported by Overholt and Rumel.[9] Only patients whom I have personally bronchoscoped are included in this report.

lary dissection and resection of the lymph nodes are a part of the standard operation for breast neoplasms. For the same reason, pneumonectomy, or removal of the entire organ, is gaining precedence over lobectomy in the treatment of pulmonary cancer. Certain benign tumors, including the so-called "benign adenomas," may be treated by subtotal pulmonary resection. Inspection of the tumor mass bronchoscopically and an actual measurement of the relative position of the tumor in respect to uninvolved bronchi is the best guide to the proper level of resection. Growths usually invade the wall of the bronchus above the point of obstruction as shown by roentgenography. Thus, it will be found in most cases that a larger rather than a smaller portion of the lung needs to be removed to excise the tumor completely.

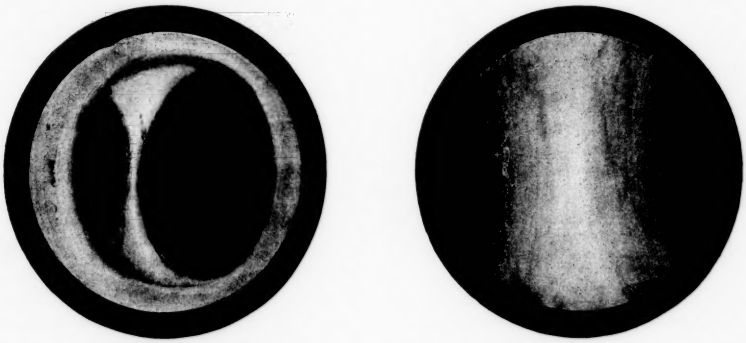


FIGURE 5. *Drawings of Normal and Widened Carinas.*

Note the narrow carina on the left, compared with the widened one on the right. The angle formed by the latter is obtuse, owing to spreading of the two main-stem bronchi by a mass of metastatic infracarinal lymph nodes. On palpating the normal carina with the tip of the bronchoscope, it is flexible and quite movable. A carina such as that illustrated on the right is hard and fixed.

Gross Pathology

Various configurations are assumed by the intrabronchial portion of pulmonary tumors. Practically all consist of both an intrabronchial and an extrabronchial segment. The semirigid cartilaginous bronchial wall undoubtedly plays a role in the configuration presented. The gross appearance of the tumor has not been found to be an accurate guide to its histologic classification, except that those tumors that are pedunculated and tend to form sessile bodies within the bronchial lumen are apt to be of low-grade malignancy, and therefore particularly suitable for surgical therapy.

Of the 45 patients who had visible endobronchial tumors on bronchoscopy, 8 presented this type of intrabronchial propagating mass. Of these, 5 tumors were found to be resectable at operation, and 4 of the 5 patients are living and well at the present time. One died suddenly, eight weeks postoperatively, after returning home. No postmortem examination was made, but clinically he had no evidence of metastatic disease. The relatively benign course of this group of 8 patients is further illustrated by the other 3, who were not resected. One had a lesion involving the carina when first bronchoscoped. This patient died twenty-five months after the onset of symptoms — about twice the average length of survival. The other 2 were explored but not resected, since involved mediastinal lymph nodes were found. One died fourteen months after onset, whereas the other is still living and in comparative good health, three years and five months after the onset of symptoms and over three years since exploration. Of the other 37 patients, there was only 1 who did not have metastases, either clinically or at exploration.

Histology

The histology of pulmonary tumors is under a great deal of discussion at the present time, and it is known that some tumors may show varying histologic patterns in different portions of the same tumor. However, the histologic classification of the biopsy specimen from a bronchus is not so important as the determination of whether

or not one is dealing with a malignant lesion. Bronchoscopic biopsy specimens are usually small, and the pathologist is not always able to classify them according to type or grade of malignancy. A diagnosis of malignancy, however, provides a satisfactory working basis on which to plan treatment.

In this series of 47 cases with positive biopsies, the pathologist in 17 cases simply made a diagnosis of carcinoma without any attempt at classification from the biopsy specimen. The others were classified as epidermoid carcinoma in 20, adenocarcinoma in 3, carcinoma simplex in 4, undifferentiated or oat-cell carcinoma in 1 and benign adenoma in 2. In 18 cases in which subsequent surgical procedures or autopsy examination provided the whole specimen, the final diagnosis was the same as the biopsy in 9, or 50 per cent.

Owing to the lack of knowledge of the histopathology of lung tumors in general and the inability to classify the tumor accurately from the biopsy specimen, it seems that for the present the treatment of all carcinomas of the lung should be the same regardless of the histology of the tumor, as revealed by bronchoscopic biopsy.

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Summary and Conclusions

Evidence points to an increase in incidence of cancer of the lung. Approximately 10 per cent of all deaths ascribed to cancer are due to primary lung tumors.

The symptomatology and physiologic disturbances in primary lung tumors are so bizarre and apparently confusing that only a complete investigation, including bronchoscopic study, provides a satisfactory basis for the determination of therapy.

Bronchoscopy is an easily performed diagnostic procedure without risk to the patient. It should be considered in every case of unexplained pulmonary symptoms.

A series of 62 verified pulmonary tumors is presented. In this group, the diagnosis was established by bronchoscopic biopsy in 46 cases, or 74 per cent.

Primary cancer of the lung should be favorable for surgical treatment in view of the high percentage of positive preoperative biopsies and the ability to make an early diagnosis by bronchoscopic means.

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AUSCULTATION OF THE HEART*

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BOSTON

The hearing ear, and the seeing eye,
The Lord hath made even both of them.
Proverbs XX: 12.

AUSCULTATION of the heart is only one of the many methods available for the study of cardiac function and for the detection of cardiac disease. Although it is well for teachers to exhort their students to learn as much as possible by other means, such as the history of the patient, inspection, palpation and percussion, there is no reason to minimize the importance of auscultation or to fail to elicit all possible aid from such a simple procedure. For among all the simple heart findings that one can make, the auscultatory observations are unique and may be regarded as *sui generis*. They cannot easily be confused with those due to other neighboring organs. Furthermore, the use of the stethoscope is easily mastered, and the instrument is available to every physician. In the present era when the economic aspect of the care of the sick is so pressing a matter, any data that can be obtained quickly and without additional cost to the patient are valuable. Moreover, there is certain information that can be obtained only by auscultation.

In performing proper auscultation, one must know what is to be heard. Gross objects are easily seen; minute findings are detected only if one searches carefully. A large goiter cannot escape detection, but a small tophus must be deliberately looked for. Likewise, a loud murmur will not be overlooked, but a telltale gallop rhythm will be heard only by the knowing ear. In a word, one must not only understand how to interpret what one hears but must also be familiar with the various possible peculiar abnormalities that are detectable by auscultation, paying particular attention to different phenomena under different circumstances. The same principle is true throughout medical practice. If a patient's primary complaint is pain in the right loin, which radiates into the groin, one should naturally search for blood in the urine, thinking of a renal calculus. Certainly one would not spend much time looking for a systolic thrill at the base of the heart. On the other hand, if a patient has attacks of syncope and there is a loud systolic murmur over the aortic area, knowing that both might be explained by the presence of aortic stenosis, one should spend a few moments, if necessary, to elicit a systolic thrill, even palpating the chest with the patient sitting up and holding a deep expiration. It is the thoughtful choice of the necessary procedures that spells success in medicine, for no physician can afford to make a so-called "complete" physical examination every time he sees a patient.

Among the various objectives in carrying out intelligent auscultation, the interpretation of the irregularities of the heart is most important. As a result of intensive study and the aid that has come from modern electrocardiography, most of the disturbances in the mechanism of the heart beat can now be detected by simple auscultation. It is a general axiom that the more thoroughly one understands electrocardiography the less one needs it. For this reason alone, it is well for students and practitioners to become familiar with this subject. It is needless in this discussion to take up all the arrhythmias, but I do want to mention some useful points in diagnosis that illustrate how sensitive the ear may be in drawing very fine conclusions from minute auscultatory abnormalities.

When the rhythm of the heart is found to be regular and the rate within normal range, one can usually assume that the mechanism is normal. Like all generalities in medicine, even this has its exceptions. If one had reason to suspect that the mechanism of the heart was disturbed, one might be led to look for some condition like auricular flutter. I recall seeing a young man who complained of palpitation. He appeared to be in good health and showed nothing abnormal on examination. The heart was regular and showed no murmur or enlargement, and the rate was 70. Directly after taking twenty-five hops, the rate was exactly 140 and perfectly regular for a minute or two, following which the rhythm became irregular and gradually returned to the previous level of 70 in a jerky fashion (Fig. 1). This simple observation led to the conclusion that the patient had auricular flutter, that the auricles were beating at a rate of 280, that at first there was a regular 4:1 block with a regular ventricular rate of 70, and that with exercise the block suddenly

*The Frank N. Wilson Lecture in Cardiology, given at Ann Arbor, Michigan, November 19, 1940.

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changed to 2:1, resulting in a regular rate of 140. It would otherwise be difficult to explain the exact doubling of the ventricular rate on effort. One might expect the rate to rise to 96, 119 or some other odd figure if the mechanism were normal. Furthermore, a normal heart would accelerate and retard gradually and smoothly following effort and would not become irregular. This simple observation illustrates how detailed a conclusion can be drawn by intelligent auscultation.

The following is another exception in which one

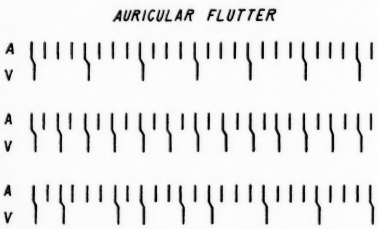

FIGURE 1. *Response to Effort in Auricular Flutter.*

The upper lines indicate the auricular contractions (inaudible), at a rate of 280 throughout; the lower lines represent the ventricular responses (audible heart rate). The first set shows a regular ventricular rate of 70; the second set, directly after brief effort, shows a regular ventricular rate of 140; the third set — a continuation of the second — shows irregular slowing to the original slow regular rate of 70.

might suspect an abnormal mechanism despite a regular normal heart rate. A forty-five-year-old woman, who had had rheumatic fever in childhood, was known to have had mitral stenosis and auricular fibrillation with a grossly irregular heart beat. She had been taking digitalis for some time, and on this particular occasion it was noted that the heart rate was 60 and perfectly regular. The question that came up was whether the auricular fibrillation had changed to normal rhythm. An early apical rumbling diastolic murmur was heard, quite typical of mitral stenosis. On careful auscultation, no murmur whatever could be detected in presystole. This led to the suspicion that auricular fibrillation was still present and that the regular ventricular rate was due to so-called "digitalis complete heart block." This suspicion was warranted, because in most cases of mitral stenosis a presystolic murmur is heard if the auricles are contracting and is absent if they are fibrillating.

When the rhythm of the heart is not regular, it is incumbent on the examiner to try to identify the exact type of irregularity. The time has passed when a physician should be content with the vague observation that the heart is "slightly irregular" or "more or less irregular" without trying to determine the type of irregularity. It is just as inadequate an observation as to say that a patient has more or less fever without determining whether it is due to typhoid fever, tuberculosis or pneumonia. A slight irregularity might be the sole clue to a diagnosis, and might lead to correct and effective therapy.

Extrasystoles are very common, occurring both with and without organic heart disease. Owing to the premature contraction of the heart, one hears a quick beat, which may or may not reach the wrist, followed by an abnormally long pause. If the extrasystole is ventricular in origin, this pause is apt to be completely compensatory so that the dominant rhythm is not disturbed; if it is auricular, the pause is not quite so long. This differentiation is not invariable, nor is it a simple matter for the ear to make this distinction. Occasionally it may be useful during auscultation to tap with one's foot in rhythm with the regular beat, not changing the pace, to see if the tap comes exactly on time or after the first beat following the pause. There is another auscultatory finding, however, that may help in distinguishing an auricular from a ventricular extrasystole. An auricular extrasystole sounds a good deal like a normal beat but is heard prematurely. The first heart sound of a ventricular extrasystole has an abnormal clicking quality. The reason for this difference in quality between the two types of premature beats is that the relation between auricular and ventricular systole is different. In the auricular, the normal sequence of auricular and ventricular contraction occurs, whereas in the other the ventricles contract before the auricles. Inasmuch as the position of the auriculoventricular valves at the moment of ventricular contraction greatly influences the quality of the heart sound, one can see that the character of the first heart sound may differ perceptibly in the two types of extrasystoles.

One may ask whether it is of any value to distinguish the various types of premature beats. Generally it is of no great moment, but occasionally it can help in identifying types of paroxysmal rapid heart action. These paroxysms come and go, and the physician may find it difficult to observe the patient while the attack is on. The finding of ventricular extrasystoles during the interparoxysmal periods may point to attacks of ventricular origin, whereas auricular extrasystoles may indicate attacks of auricular origin. Obviously, prognosis and treatment may be quite different in the two conditions.

The diagnosis of auricular flutter is generally

very difficult without electrocardiographic aid. In many cases, however, with increasing experience and study, it has become possible to make accurate bedside diagnoses by auscultation. One simple point briefly mentioned above deserves further emphasis. It should be recalled that the sounds result mainly from ventricular contraction. One thereby infers what the auricles are doing. In flutter, the auricles are contracting very rapidly, perfectly regularly and inaudibly. The audible ventricular rate is slower, and may be regular or irregular. When it is regular, the rate must be a simple fraction of the auricular rate (except for the rare cases in which there is a 1:1 response). This means that if the heart rate is 140 the auricular rate must be 280. It cannot be 265 or 311 or any other figure. If the regular ventricular rate is 168, the auricular rate must be 336. Hence, realizing that the auricular rate in an untreated case of flutter is hardly ever under 200 or over 360 and that there is practically always an accompanying heart block, one can draw some simple conclusions. If a regular heat rate is about 198, it cannot be due to flutter, for the auricular rate would have to be 396, which is too rapid or 198, which is too slow. The same reasoning applies to ventricular rates around 210 to 230.

There is another peculiarity that is quite characteristic of auricular flutter. When the mechanism of a regular rapid heart is unknown, vagal

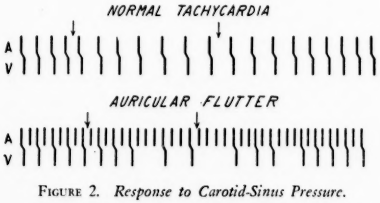

FIGURE 2. *Response to Carotid-Sinus Pressure.*

A represents the auricular beats; V, the ventricular beats. The arrows indicate the duration of carotid-sinus pressure. The upper tracing shows gradual slowing from vagal stimulation and a smooth return to the fast rate in a case of normal tachycardia. The lower drawing shows sudden slowing and a jerky return to the original fast rate in a case of auricular flutter. Both started and ended with a regular rate of about 150.

slowing by carotid-sinus pressure may help to reveal the underlying disturbance. Everyone knows that if the attack is abruptly stopped by such a procedure the condition must have been a paroxysm of auricular tachycardia, for no other mechanism will respond in such a fashion. However, if there is temporary slowing with prompt gradual return of the rapid rate, one can be quite sure that it is not a paroxysm of auricular tachycardia, but there is still doubt whether it is a normal sinus tachycardia or auricular flutter. If careful attention is paid to the way in which the previously rapid rate is resumed after the temporary slowing takes place, this differentiation can be made (Fig. 2). In normal sinus tachycardia, the process is smooth and gradual, each beat becoming faster and faster until the original rapid rate returns. In flutter, on the other hand, the recovery of the tachycardia occurs in an irregular or jerky fashion. After the temporary pause or slowing, one or two quick beats may occur, then a slow beat, another quicker beat followed by a somewhat slower one,

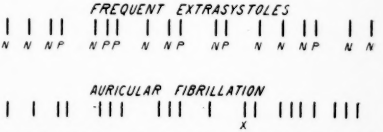

FIGURE 3. *Difference between Frequent Extrasystoles and Auricular Fibrillation.*

The upper drawing shows many premature beats (P) preceded by normal beats (N) and followed by compensatory pauses. The lower drawing, which superficially resembles the upper, also shows quick and slow beats, but at one point (X) a slow beat is not preceded by a quick beat. This typifies auricular fibrillation.

until finally the original rapid regular rhythm is resumed. These irregular beats are heard because while the auricles have been very rapid, regular and entirely undisturbed by the vagal stimulation, the degree of auriculoventricular block has been varying. Similar observations may be made if the patient can hold a deep breath or is asked to perform some brief effort. These minute auscultatory changes enable the physician to draw very accurate observations at the bedside.

After a certain amount of training and experience, the diagnosis of auricular fibrillation by auscultation in most cases becomes a fairly simple matter. Occasionally difficulties arise, for it may be confused with auricular flutter when there is a changing degree of block, with frequent extrasystoles or other less common combinations of arrhythmias. In some cases, there is no way of establishing a certain diagnosis without electrocardiographic study. Frequently, however, when confusion arises, correct decisions can be made by the use of simple methods. When for one reason or another the possibility of auricular flutter arises, accelerating the heart rate by having the patient bend up and down in bed several times will give

the answer. In flutter, the rate is apt to become rapid and perfectly regular for a short while, and in fibrillation the gross irregularity will persist. What is more difficult is to distinguish frequent and multiple extrasystoles from auricular fibrillation. Both conditions may have short and long pauses between beats. There may be short runs

FIGURE 4. *Difference between Paroxysmal Auricular and Paroxysmal Ventricular Tachycardia.*

The upper drawing shows perfectly regular beats in a case of paroxysmal auricular tachycardia, at a rate of 180. The lower drawing shows slight but definite irregularities in a case of paroxysmal ventricular tachycardia, at the same rate.

of quick beats followed by pauses when there are two or more extrasystoles in quick succession or when they are interpolated. Both may have an appreciable pulse deficit, and may appear to present a total irregularity of the heart. The finding that identifies the arrhythmia as auricular fibrillation is the occurrence of a pause not preceded by a quick beat (Fig. 3). A sudden lengthening of the cycle that follows a quick beat may be due to a compensatory pause after an extrasystole or

FIGURE 5. *Changing Quality of Sounds in Ventricular Tachycardia.*

The upper lines represent auricular contractions, at a rate of 113; the lower, the ventricular contractions, at a rate of 190. Note the changing time relation between the auricular and ventricular contractions; this changes the quality of the first heart sound.

to fibrillation, but a sudden pause following a beat of normal length will for the most part be due only to auricular fibrillation. After hearing such a sudden lengthening of the heart cycle, one must think back and recollect whether or not the previous cycle was short.

There are characteristic auscultatory findings in most cases of ventricular tachycardia, one of the uncommon but very important cardiac arrhythmias. Unlike paroxysmal auricular tachycardia, in which the heart beat is almost invariably perfectly regular and the consecutive sounds of identical character, this disturbance is frequently slightly irregular and there may be slight but definite alterations in the intensity or quality of the first heart sound (Figs. 4 and 5). For ten or more beats, the rhythm may be perfectly regular and the heart sounds quite similar in character, just as in auricular tachycardia, and then suddenly for two or three cycles slight but detectable irregularities appear both in time and in the quality of the beats. This peculiarity is due to the changing relation between auricular and ventricular systole that takes place in many cases of ventricular tachycardia; this relation is constant in auricular tachycardia. On rare occasions, I have detected this peculiar alteration in the quality of the heart sounds even when the ventricular rate was perfectly regular, because the auricles were contracting independently and at a different rate. These detailed observations can be made at the bedside by any well-trained physician and may enable him to arrive at a quick decision and institute proper treatment. From a therapeutic point of view, the decision may be a matter of life and death, for ventricular tachycardia generally responds favorably to quinidine therapy and is refractory or aggravated by digitalis.

The diagnosis of the different degrees of heart block can often be recognized by auscultations. In first-degree heart block (simple delayed PR interval), the rhythm of the heart is perfectly regular. This disturbance, therefore, is generally overlooked, unless graphic registration of the heart beat is employed. However, if there is some reason to suspect its presence, as during rheumatic fever, the detection of a definite diminution in the intensity of the first heart sound at the apex points more strongly to this diagnosis. This is particularly true if the intensity of the first heart sound is observed to diminish during the illness. The reversal of this process can also be followed, that is, the gradual return of the previous normal intensity of the first heart sound as the patient is recovering and the conduction time is becoming normal. The reason for the above phenomenon is that the loudness of the first sound is directly related to the PR interval. It is loudest when it is between 0.04 and 0.10 second, normal in intensity between 0.12 and 0.16 second, and gradually becomes fainter when it is 0.20 to 0.28 second or longer. These differences, as mentioned above, probably result because the mitral and tricuspid valves are caught in different positions at the moment of ventricular systole, depending on the time the auricles contract.

Occasionally, in first-degree heart block, one may hear a third heart sound in diastole, resulting in what sounds like a gallop rhythm. This is probably in some way related to the early contraction of the auricle. When it is present and the commoner causes of gallop rhythm, such as hypertensive heart disease and coronary-artery disease, can be satisfactorily dismissed from consideration, it should make one suspect delayed conduction time. These auscultatory findings are of particular importance because there are no other simple means of detecting this condition, and the fact that the heart is perfectly regular otherwise makes the physician entirely oblivious to

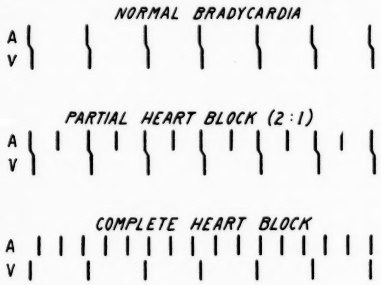

FIGURE 6. *Bradycardia.*

The first drawing (normal bradycardia) shows auricles and ventricles beating slowly and regularly, at a rate of 35. The second (2:1 heart block) shows an auricular rate of 70 and a ventricular rate of 35. The third (complete heart block) shows an auricular rate of 93 and a ventricular rate of 35. In all, there is a regular ventricular rate of 35, but only in the last is there a changing quality of the first heart sound, because of changing relation between the auricular and ventricular systoles.

any disturbance of the mechanism of the heart beat.

Second-degree or partial heart block (dropped beats) is readily recognized because the loss of an entire heart cycle is easily heard on auscultation. It must not be expected that the pause will be quite so long as two normal heart cycles, because the last beat before the pause is accompanied by a long PR interval and the one after the pause by a much shorter PR interval. In sinoauricular block, a far rarer condition, this pause is much likelier to be exactly equal to two heart cycles. Partial heart block must not be confused with extrasystoles. In both, the peripheral pulse may intermit for an entire cycle, but with extrasystoles a premature beat ought to be heard over the precordium during this interval, and in block no such beat will be present. The difficulty is that premature beats are very rarely inaudible and in auriculoventricular block a faint auricular sound may resemble an extrasystole. In these cases, one is entirely dependent on electrocardiography.

There is one auscultatory sign that is quite pathognomonic of complete heart block. Apart from the slow, generally regular beat of 30 to 40, one should pay particular attention to the quality of the first heart sound in different cycles. If the heart is slow at a rate of about 35 to 40 and regular, it can be due to complete block, to a regularly recurring 2:1 block or even to a normal bradycardia (Fig. 6). In the last, there may even be no heart disease whatever. The differentiation between these conditions may therefore be quite important. If there is complete dissociation (except for the rare cases in which auricular fibrillation is also present), the first heart sound varies in intensity or quality in different cycles, sometimes suddenly becoming booming or muffled or reduplicated. Just as in ventricular tachycardia, this is owing to the changing relation between the time of auricular and ventricular systole. When the slow rate is due to 2:1 block or to a normal bradycardia, the sounds are all alike. It must be borne in mind that hearing auricular beats during the long ventricular pauses, although a common finding in heart block, does not mean that the block is complete. It is the peculiarity of the first heart sound above mentioned that identifies the condition as complete dissociation.

Apart from the irregularities of the heart, the quality of the sounds may have significance. Distant heart sounds need not indicate heart disease, since they often result from pulmonary emphysema or a thick chest wall. In the absence of these or other obvious causes, and especially if the heart sounds are known to have been of normal intensity shortly before, a marked diminution in the intensity of both heart sounds occurs in some cases of acute coronary thrombosis and in extensive pericardial effusion. Apart from these considerations, so-called "weak heart sounds" have little clinical value. The term is often misleading, for the connotation that the heart is weak need not be true.

Accentuation of the first heart sound may have considerable diagnostic value. It is frequently an early evidence of developing mitral stenosis and should lead the examiner to search with greater care for the short presystolic or mid-diastolic murmur that characterizes mitral stenosis. Furthermore, a snapping first heart sound may be the first clue that the patient has hyperthyroidism. It is obvious that in the classic case of thyrotoxicosis

other features, like exophthalmos, enlarged thyroid gland, a typical history of nervousness, palpitation and loss of appetite, readily reveal the true nature of the underlying morbid process. In some of the obscure cases, however, many of these points are lacking, and one may be led to the diagnosis by auscultation of the heart. Accentuation of the first heart sound may also be found in nervous patients, in hypertension, in anemia and in some fevers, especially rheumatic fever.

There is one condition that I especially wish to mention because it has only recently been recognized as a clinical entity and is one in which the first sound is also distinctly accentuated. It is the syndrome of short PR interval with curves resembling bundle-branch block occurring in patients prone to have paroxysms of rapid heart action. It was first called to attention as a clinical unit by Wolff, Parkinson and White,[1] although Wilson[2] reported an isolated case of this sort many years before. These patients, who apparently show no evidence of organic heart disease, are subject to attacks of palpitation and show an unusually short PR interval when the heart rate and rhythm are normal. The only method suggesting the presence of the abnormal mechanism before electrocardiograms are taken is the detection of an unusually accentuated first heart sound. I mention this because only recently I had such an experience, and the auscultatory suspicion was validated by electrocardiographic study.

In the diagnosis of acute pericarditis, one is almost entirely dependent on auscultation. There is nothing in the history or in other methods of examination that is so helpful diagnostically or in fact that may throw any light whatever on the diagnosis. Unless one hears the telltale to-and-fro pericardial friction rub, one is generally unable to make the diagnosis of acute pericarditis. Inasmuch as this sign is likely to be transient and may last only a day or so, frequent auscultation is necessary. Occasionally, it is heard only if the patient sits up or bends forward. Apart from its specific diagnostic value, its detection may help in identifying one illness as being rheumatic, another as acute coronary thrombosis, and so on.

Gallop rhythm is a purely auscultatory phenomenon. Although in some cases there are other associated phenomena, such as bundle-branch block, a doubling of the apex impulse and pulsus alternans, the quality of the heart sounds alone has a specific connotation. Care must be taken to differentiate a diastolic from a systolic gallop. The former almost invariably signifies grave heart disease; the latter is found in structurally normal hearts. The differentiation may be facilitated by the following procedure. When a so-called "normal" or midsystolic gallop is present, it is heard at the apex, and on rhythmically moving the stethoscope toward the base of the heart, one finds that the abnormal third sound gradually disappears. It should also be noted that the sound to disappear is the middle one of the three, leaving the two normal heart sounds that are customarily heard at the base of the heart. It is also of some clinical interest that the common diastolic gallop rhythm that generally indicates a grave prognosis is extremely rare in auricular fibrillation or flutter. Occasionally, this antagonism is indirectly helpful in diagnosis, when a gallop disappears and a new rhythm is established. Finally, a word of caution may be appropriate concerning the confusion between cases with gallop rhythm and those showing a normal third heart sound or those in which a third sound in mid-diastole proves to be the early evidence of developing mitral stenosis. Experience and mature judgment are required to make these differentiations.

Pulsus alternans is a peculiarity of the peripheral pulse in which alternate regular beats are strong and weak. It connotes grave heart-muscle disease. Not infrequently, this may be detected on auscultation of the heart itself, when the heart sounds or an accompanying systolic murmur are found to alternate in intensity.

There are other extremely rare cases in which peculiar sounds are heard over the precordium. A tinkling or splashing sound with each heart beat suggests both air and fluid in the pericardial sac or a diaphragmatic hernia with the heart pulsating against the stomach contents. Recently Hamman[3] described unusual crunching sounds with the heart beats in acute mediastinal emphysema, a condition that is easily confused with acute coronary thrombosis. These are all very rare conditions, but they are recognized mainly through auscultation.

Finally, the subject of cardiac murmurs must be considered. This is not the occasion to discuss the significance or interpretation of specific murmurs. I do wish to emphasize that the detection and proper interpretation of murmurs is a very important function of the clinician. It is true that the question whether a heart is failing is not dependent on the presence or absence of murmurs. Nor may the presence or absence of murmurs decide whether the symptoms of which a patient complains are due to the heart. But very often the pres-

ence or absence of murmurs determines whether there is organic heart disease. No simple method of diagnosis other than auscultation is valid in chronic valvular disease. Very often, the decision whether the heart is perfectly normal or whether there is early aortic insufficiency or mitral stenosis can be made only when a faint diastolic blow along the left sternal border or a short presystolic murmur at the apical region is heard. In some cases, the diagnosis depends entirely on these auscultatory findings, for electrocardiograms and roentgenograms may fail to reveal significant abnormalities. When it is realized that patients have compensated organic valvular disease for many more years than they have heart failure and that they often desire to know whether heart disease of any sort exists, this subject must be of considerable importance.

As a corollary of the above, it is well for physicians to develop confidence in detecting cardiac murmurs, for if one can be certain that there are no murmurs whatever it makes it extremely unlikely that there is any valvular disease. This question is of significance for patients who have recovered from rheumatic fever or chorea. When they are seen years later, the complete absence of murmurs means that the heart has entirely escaped permanent injury and they may be regarded as normal, unless a new infection later develops. While any murmur is present, although they may yet carry on normally, they are subject to the hazard of progressive stenosis of the valves and subsequent congestive failure or superimposed bacterial endocarditis. In other words, it is my opinion that apart from the acute stages of rheumatic infection, when the involvement of the heart muscle is paramount, the subsequent health of the patient depends entirely on involvement of the valves. The development of chronic heart failure in rheumatic valvular disease is directly or indirectly the result of the mechanical insufficiency or constriction of the valves and is not dependent on any peculiarity of the heart muscle. It may be considered that the myocardium recovers completely or that at least its involvement is insignificant in determining the ultimate outcome in most patients with rheumatic heart disease who do not succumb early, during the active stage of infection. This point of view is emphasized to counteract a prevailing concept that places all importance on the myocardium in determining the development of heart failure. Although the myocardium is of primary significance in hypertensive heart disease and in coronary-artery disease, I believe that it plays a minor role in chronic rheumatic valvular disease.

There are many more valuable diagnostic points to be elicited by auscultation than I have mentioned. Some have been deliberately omitted because they are common knowledge, others because of my own limitations. There remains much that will yet be learned by further study. If I appear to have overemphasized the use of auscultation, it must be laid to my interest in this subject. It has one great advantage, however: the procedure is valuable and simple, and the data are inexpensively obtained and available to all students and practitioners. If for no other reason, it merits serious consideration.

270 Commonwealth Avenue

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POLLEN AND MOLD SURVEY OF SOUTHEASTERN NEW ENGLAND—1940

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THERE are few reports on the seasonal contamination of the air in New England with the pollens and molds that are proved excitants of asthma and hay fever. Rackemann and Smith,[1] Durham[2-4] and Pratt[5, 6] have each recorded pollen or mold surveys from single stations in Boston. Sylvester and Durham[7] have presented a relatively complete study of the ragweed season in Maine, demonstrating the diminishing amount of ragweed pollen found in the air as one progresses north from Portland. In view of the paucity of such reports, it was considered advisable to make a more thorough co-operative study, with special reference to the variation of daily counts from adjacent stations within the City of Boston, and to compare these with counts at Worcester, Providence and suburban Boston.

METHOD

Six stations were established, brief descriptions of which follow:

Station 1: Boston. The roof of the five-story main building of the Children's Hospital, Longwood Avenue, exposed to the wind from all directions.

Station 2: Boston. A third-floor window frame at the Beth Israel Hospital, Brookline Avenue, exposed to the wind from the northeast, east and southeast.

Station 3: Boston. The roof of the five-story Lahey Clinic building on Commonwealth Avenue, exposed to the wind from all directions, but especially from the east and west.

Station 4: Newton Center. Six inches above the ground, 25 feet from a house in a suburban district where the houses are 50 to 100 feet from each other, exposed to the wind from all directions, but especially from the southwest, southeast and northwest.

Station 5: Providence, Rhode Island. A second-story porch in the residential district, exposed to the wind from the northeast, east, southeast and south.

Station 6: Worcester, Massachusetts. The roof of the three-story Memorial Hospital in a thickly settled district, exposed to the wind from all directions.

Stations 1, 2 and 3 are on the western edge of Boston and within a mile of each other. The area is characterized by apartment houses, vacant lots and shade trees.

At each of the six stations, slides were exposed under a cover 2 to 3 feet square and 6 to 8 inches above the slide. The sides were open to the weather. Between 8 and 9 o'clock each morning, a slide coated with glycerin jelly was exposed at each station and collected twenty-four hours later. Over week ends and on holidays, slides were exposed for forty-eight hours. The pollen grains and Alternaria spores falling on 1.8 square centimeters were identified and counted. The slides were stained with Calberla's fluid to assist in identification of the pollen grains. In addition, the number of colonies of Alternaria growing on a thin layer of Sabouraud's medium in Petri dishes exposed to the outside air for a half hour three times weekly was determined. Taking into account time and area, the slide counts should theoretically be about 40 per cent higher than the plate counts.

Charts were constructed in which the number of pollen grains or Alternaria spores falling on 1.8 square centimeters in twenty-four hours is represented as the ordinate and the dates as the abscissa. Where slides were exposed for forty-eight hours, the counts were divided by two and charted as if for individual days.

RESULTS AND COMMENT

Figures 1, 2, 3 and 4 represent the pollen counts for the average of the three Boston stations and for Newton Center, Worcester and Providence respectively. All show the same variations with fairly well-demarcated tree, grass and weed seasons. Table 1 itemizes the actual dates when appreciable amounts of pollen were in the air at each station for the 1940 season. It will be observed that the dates are remarkably similar for each station except for the end of the grass season, which terminated gradually during July. The weed pollens stopped abruptly with frost on September 24.

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A comparison of Figure 1 for the Boston stations and Figure 2 for Newton Center demonstrates the difference between city and suburban areas. In both places, the tree counts are of the same order of magnitude, because shade trees are numerous in the parts of the city studied. However, it is not surprising to find that grass and weed pollens were much more abundant in the suburban area.

Figure 5 represents the pollen counts at two Boston stations only a few hundred yards apart. Failure of the pollen peaks to be consistently superimposed demonstrates that local variations in vegetation and wind currents may grossly affect

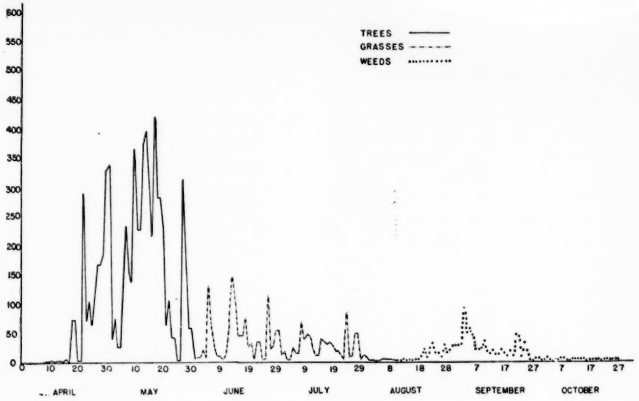

FIGURE 1. *Average Pollen Counts at Three Boston Stations.*

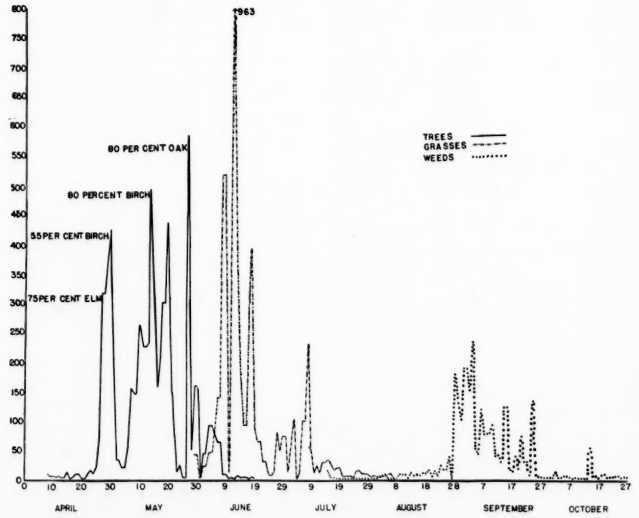

FIGURE 2. *Average Pollen Counts at Newton Center.*

the counts. This suggests one reason why all hay-fever patients do not have severe symptoms on the same days.

and asthma, and yet when pollen counts are made by the gravity method, the weeds appear to be the least important, if quantity is any criterion.

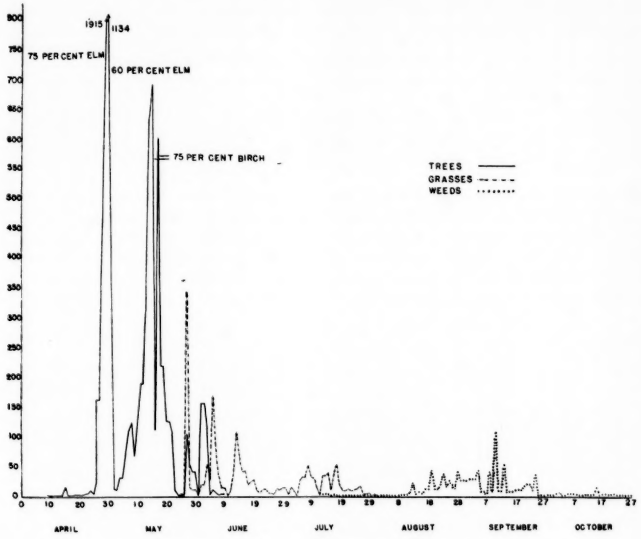

FIGURE 3. *Average Pollen Counts at Worcester.*

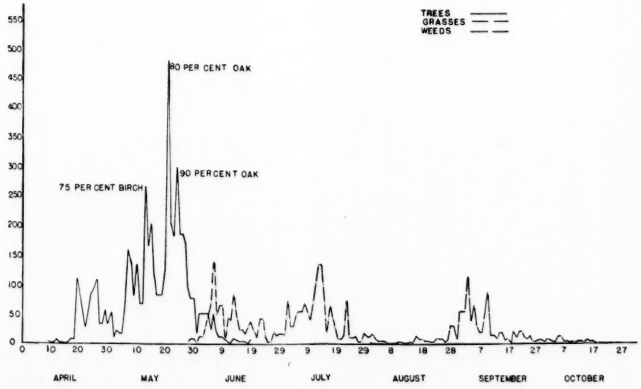

FIGURE 4. *Average Pollen Counts at Providence.*

It has been recognized from clinical evidence that the weed pollens from late August to frost are the commonest seasonal excitants of hay fever

This paradox is explained when the number of pollen grains per cubic yard of air is calculated. Stokes's law for falling bodies, as adapted by

Scheppegrell[8] and corrected by Cocke,[9] has been experimentally shown by Cocke[10] to be accurate for pollen grains if the spiculated surface of the weed pollens is taken into account. Utilizing Cocke's tables and assuming that Wodehouse's[11] figures for pollen size are correct, the average number of pollen grains per cubic yard of air for each day was calculated for Newton Center and is shown in Figure 6. Because of its small size and spiculated surface, the ragweed granule settles out of the air much more slowly than the larger, smooth, tree and grass grains. Figure 6 gives a clearer picture of the clinical significance of the three pollen seasons. Taking thirty-one consecutive days in each of the three seasons and calculating the average contamination of the air per cubic yard for this period for each season, the results are: trees 415, grasses 250 and weeds 555.

Among the trees, elm, birch and oak were by far the most abundant pollinators and are therefore probably the most important clinically. There were moderate amounts of poplar and maple pollen and a little alder, ash and beech. Pine pollen frequently appeared on the slides in fairly large amounts, but since pine pollen contains no atopic excitant,[12] it was omitted from the charts and calculations. At two stations where linden trees were in the neighborhood, a small quantity of pollen was recovered in July, but linden is a flowering tree and is not generally considered to be a cause of hay fever or asthma.[11] It, too, was omitted from the charts. Occasional grains of unidentified pollen were also disregarded.

There are five common grasses in New England: June, orchard, sweet vernal, red top and timothy. The pollens of these grasses and of plantain are very difficult to differentiate, and therefore no effort was made to separate them. Furthermore Rackemann and Wagner[13] have shown by desensitization of passively sensitized skin sites

TABLE 1. *Pollens During 1940 Season.*

STATION	TREES	GRASSES	WEEDS
Three Boston stations	April 22–May 27	June 5–late July	August 20–September 24
Newton Center	April 27–May 30	June 6–July 8	August 26–September 24
Worcester	April 26–May 22	May 27–late July	August 19–September 24
Providence	April 19–May 28	June 5–July 23	August 28–September 10

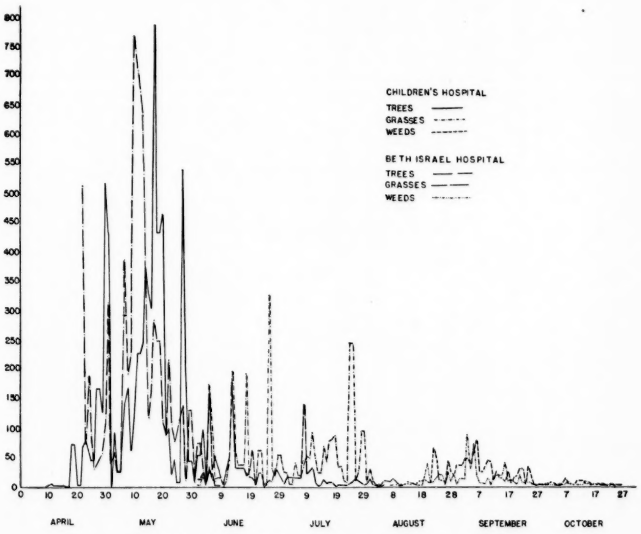

FIGURE 5. *Average Pollen Counts at Two Boston Stations.*

that although no one of the three commonest grasses (timothy, orchard and red top) invariably desensitizes to the two others, crossed reactions out the clinical observation of the overwhelming consequence of ragweed in fall hay fever.

Among the mold spores producing respiratory

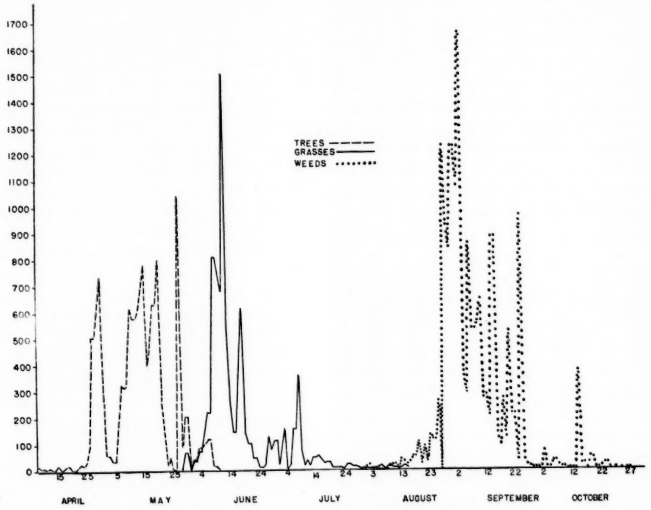

FIGURE 6. *Average Pollen Counts at Newton Center.*

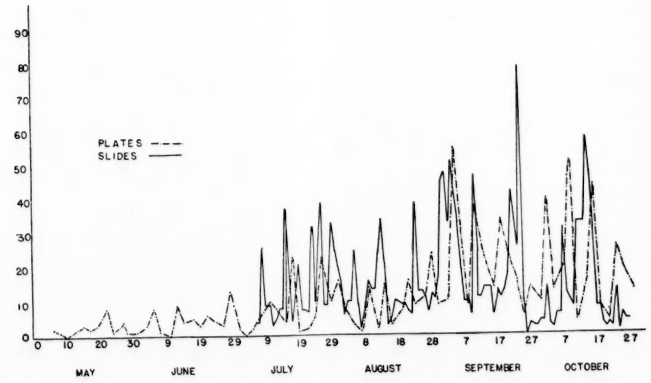

FIGURE 7. *Average Alternaria Counts at Two Boston Stations, Newton and Worcester.*

between the different species are very common.

The weed pollens consisted of ragweed (92 per cent), goldenrod (4 per cent), cocklebur (3.2 per cent) and wormwood (0.8 per cent). This bears allergy, Alternaria is the commonest.[8] Figure 7 summarizes the results for the four stations at which exposures of slides and plates were made regularly (two Boston stations, Newton Center

and Worcester). To produce more consistent curves, the chart was constructed by adding the daily slide counts of the four stations and graphing the daily total as a solid line. The sum of the plate counts is represented as an interrupted line. Therefore, the curves are four times higher than they would be for a single station. Although the two curves do not superimpose, they are both of the same order of magnitude, the slide counts averaging slightly higher, and show the same seasonal fluctuation. The total counts for the season from the four stations gave essentially the same results except that the total for Newton Center was somewhat greater than that for the city.

The Alternaria season does not have such definite time limits as the pollen seasons. As shown previously,[5, 6] Alternaria can be cultured from the air in Boston throughout the entire year, but the heaviest contamination comes with the onset of warm weather, in July, when the grass counts are dwindling. The season reaches its peak with the weeds but persists into November long after ragweed pollen has left the air.

It will be noted that the counts of Alternaria here recorded are low compared with the pollen counts. However, the spores probably settle out of the air considerably more slowly than pollen because they are smaller and irregular in shape. Stokes's law does not apply to the calculation of their rate of fall, since they are not spherical. It seems probable that the per cubic yard contamination of the air with Alternaria spores is not much less than that of ragweed pollen in late August and early September.

Summary

A survey of the pollens producing hay fever in southeastern New England shows three clearly defined seasons: trees from April 19 to May 30, grasses from early June to mid-July, and weeds from August 20 to late September. The Alternaria spore season overlaps the grass and weed seasons and persists later into the autumn.

Grass and weed pollens are more abundant in suburban areas than in the city, whereas tree pollen is equally plentiful in town and city because of the numerous shade trees in the areas of the city studied.

Failure of pollen peaks from neighboring stations to be superimposed consistently demonstrates the effect of local vegetation and wind currents.

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MEDICAL PROGRESS

PHYSICAL THERAPY*

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BOSTON

ONE of the outstanding events in physical medicine during the last two years was the publication of an excellent textbook by Krusen.[1] There has long been a need for a reference book to which the student or practicing physician could turn for sound and detailed information on the practical aspects of physical therapeutics. Krusen is an authority on the subject, and has had unusually large experience in clinical application and investigative work. In addition to comprehensive presentations of the subjects of heat, light and electrotherapy, he has included the technic and indications for the use of hydrotherapy, massage and exercise — measures that have received little acclaim recently because of enthusiasm concerning electrical apparatus, which actually have only a small role in physical therapy. The present knowledge of the physical and physiologic principles involved has been summarized, with extensive references. The illustrations and explanations of simple measures that may be used in the home are especially valuable.

Hypothermia has recently been found to have more new useful applications than any other physical measure. Many investigative studies have dealt with the physiologic changes and therapeutic effects of the reduction of general body temperature and the local use of cold. The present status of this therapy has been reviewed by Talbott.[2]

Recent developments in the use of the majority of other physical agents are presented in relation to the different medical specialties.

MEDICINE

In the treatment of rheumatoid arthritis and degenerative joint disease, physical measures continue to have a significant role. In these conditions with a tendency to chronicity, simple procedures that can be carried out in the home should be emphasized. Krusen[3] has outlined specific instructions for the use of homemade bakers, clamp lamps, hot paraffin packs, whirpool baths and contrast baths. Massage, when skillfully administered on nonacute joints, is beneficial, and a lay person with proper instruction may learn simple technics that are of value in improving local circulation and in relieving muscle spasm. It is recognized that training in body mechanics is effective in preventing undue strain and trauma on diseased joints.[4] Postural and specific graded exercises for increasing joint range and muscle power are probably of more benefit than any other physical measure when properly supervised.[5] Diathermy,[6] cabinet baths and more elaborate types of treatment have a place in hospital departments.[7] However, results of fever therapy in rheumatoid arthritis and osteoarthritis have been disappointing.[8] Iontophoresis with histamine and acetyl-beta-methylcholine chloride has given encouraging results,[9–12] but has not replaced other simpler agents for symptomatic relief. Neurologic symptoms may accompany cervical degenerative arthritis, and much benefit may be obtained by heat, massage, traction, manipulation and posture training.[13] The temporomandibular joint is frequently involved and often is not treated adequately. Bayles and Russell[14] brought out the necessity of complete rest and local heat during the acute stages, with mild gum-chewing exercises later. Coccygodynia, when caused by spasm of the levator ani, coccygeus and piriformis muscles, has frequently been relieved by internal massage, according to the experience of Krusen and Basom.[15] They also reported success from firm massage over the fibrous nodules, in addition to local heat and stretching exercises, in the treatment of fibrositis.

Short-wave diathermy has been reported as curing lung abscess in 85 of 128 cases in the literature, as reviewed by Brugsch and Pratt.[16] In 8 cases, however, applications for ten to forty-five minutes were given without effecting a cure. Only 2 of these cases were acute, and this method of treatment may deserve further trial in the acute stage. In spite of the steadily improving results of chemotherapy for pneumonia, diathermy is still thought to be a valuable adjunct in treatment, since it decreases cyanosis by improving pulmonary circulation.[17] Overdosage is to be avoided because of the possible edema.

In the management of cardiac conditions during convalescence, a carefully supervised, indi-

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vidual program of physical measures is beneficial.[18] This may consist, at first, in gentle stroking and passive motion of the extremities, followed later by graded active exercise to improve peripheral circulatory efficiency.

General Surgery

The aim of physical therapy in traumatic conditions is to shorten convalescence and rehabilitate the patient. In fractures, the early use of measures to soften or eliminate induration, diminish swelling, and lessen redness and heat ensures early function.[19] Elevation, gentle stroking, sedative massage, prolonged mild heat and assisted active exercise without pain may accomplish this.[20–24] The frequency of treatments is of some economic importance. Butler[25] has recommended daily treatments so long as there is progress, as measured by careful observations. Occupational therapy may then take over and speed return to work. Contusions of muscles may lead to myositis ossificans and prolonged disability. Preventive measures should be used in the early stages to control hemorrhage by cold applications and a compression bandage; massage on severe tender contusions should be avoided. Later, heat aids in the absorption of the hematoma.[26] Injuries involving tendons and nerves, particularly of the hand, should be splinted in a position of physiologic rest, care being taken to avoid tension on sutures.[27] Experimental studies of the tensile strength of healing tendons have revealed that motion should not be started until after sixteen days; it should then be restricted for two weeks more, when free active motion is safe.[28]

In the treatment of peripheral vascular disease, the value of alternate positive and negative pressures, by means of a leg boot and intermittent venous occlusion, appears to be limited.[29] The careless use of local heat, particularly diathermy, is dangerous, since the metabolic requirements may be increased beyond the circulatory capacity and gangrene may develop.[30] When heat is indicated, either a thermoregulated cradle or reflex heating by warming the upper extremities or by applying diathermy over the sacral areas is desirable.[31, 32] Iontophoresis with acetyl-beta-methylcholine chloride does not replace surgical measures for varicose and post-thrombophlebitic ulcers, but after simpler methods have failed, some discomfort may be relieved by this means and healing promoted.[33, 34] Buerger's exercises continue to be a valuable adjunct in treatment when carefully prescribed,[35] and the oscillating bed tends to effect the same results when active exercises are impossible.[36]

Since the advent of extensive chemotherapy, local measures have assumed less significance in the treatment of genitourinary infections. Diathermy, however, is still useful for symptomatic improvement in chronic prostatitis and pelvic inflammatory disease.[37]

A major part of treatment following thoracoplasty is the prevention of scoliosis and limited painful motion of the shoulder girdle and arm. A carefully graded set of exercises with skillful supervision gives gratifying results.[38]

The sterilization of air in the operating room by means of ultraviolet radiation has been studied, and methods have been developed whereby it is claimed that contamination from unsterile air can be practically eliminated.[39–42] Similar methods have been valuable in reducing the incidence of respiratory-tract infections on hospital wards.[43–45]

Orthopedic Surgery

The problem of chronic backache and sciatic pain has not been solved, as indicated by the numerous publications recommending different treatments. A recent symposium[46] on diagnosis and treatment has been published and contains much valuable information concerning the relation to backache of posture, visceral disease, fractures and other trauma, faulty mechanics and inflammatory processes. Surgical and conservative measures in treatment are discussed. Kuhns[47] reports the relief of sciatic pain and low-back disability in 771 of 1000 cases by means of rest on a flat bed with hips flexed for ten days and daily application of heat, followed by graded exercises and support when the patient is ambulatory. The different physical measures indicated in backache from various causes include traction, manipulation, special stretching and postural exercises, massage, particularly firm massage in fibrositis, and local application of heat.[48] Corsets are frequently helpful, and the proper types to be used have been discussed by Ober.[49]

A specific exercise program is of distinct value in the treatment of scoliosis. Colonna and vom Saal[50] found that 30 per cent of 500 cases of poliomyelitis developed scoliosis. The presence of asymmetrical paralysis and inadequate support were important factors in its development. Muscle-training and chest-expansion exercises were helpful, but spinal fusion was usually indicated in the presence of moderate and severe deformity. Conservative treatment in carefully selected cases has been successful in Steindler's[51] experience. The lumbar and cervicothoracic sections of the spine were limbered up to promote derotation and the development of countercurves to establish compensation. Adequate musculature was essential for success. In those cases requiring fusion, the

physical therapeutists are called on for special limbering and manipulative treatments prior to operation.[52, 53]

In deformity of the back due to juvenile kyphosis, physical measures are relied on almost exclusively in treatment. These consist in support, rest, local heat, light relaxing massage and corrective exercises of graded intensity.[54] Wheeldon[55] has suggested that contracted hamstring muscles may have a role in the production of poor posture and juvenile kyphosis. Fifty per cent of his cases were improved by passive hamstring stretching and remained improved. Twenty-five per cent tended to recur when the exercises were stopped. The remainder were unaffected by exercises. In this last group, he has had success by resection of the hamstring muscles.

Orthopedists and physical therapists have frequently expressed the advantages of good posture. Joplin[56] has applied the principles of posture in occupational therapy, which is a timely and worthwhile contribution. Most of the concepts of postural instruction are based on empirical evidence. A metabolic study, however, showed that the energy expenditure of standing was very small, and the sense of fatigue associated with motionless vertical posture may be due to cerebral anemia, which can be overcome by sway and relaxed stance.[57]

The chief development in the treatment of infantile paralysis that commands interest at present is the so-called "Kenny method." Briefly, this consists in nearly continuous daily hot fomentations during the acute stages to relax spasm, no splinting, early passive and active exercises without pain, and special muscle re-education, concentrating on the insertion of muscles, with attention to body mechanics. Kenny's theory is that the important features of acute poliomyelitis are muscle spasm, inco-ordination due to pain and substitution of muscle function, and mental alienation, because of which the patient forgets normal muscle performance or loses the idea of making muscles contract. Complete evaluation of this treatment is not possible, according to preliminary reports.[58] Certainly, quick recoveries have been observed, but it is too early to determine end results. Lewin[59] has written a splendid book covering the many aspects of diagnosis and treatment of this disease.

The treatment of painful shoulders caused by bursitis is still unsatisfactory in spite of reports of success with novocain injections, saline irrigations, operative removal of calcium deposits, manipulation, rest, local heat, exercise and large doses of ammonium chloride.[60–67] A carefully controlled study of these various methods is needed for proper evaluation.

Foot disabilities are common and are frequently due to mechanical strain. Training in proper use of the feet, special foot exercises, posture correction, contrast baths and proper shoes are measures recommended in prevention and treatment.[68, 69]

Neurology and Psychiatry

The most interesting recent development in the treatment of mental disease by physical agents is the use of electric-shock therapy. The first clinical work was done in the Rome clinic by Bini[70] in 1938 and by others.[71] The advantages claimed for this method over other types of shock therapy are simplicity, as compared with intravenous medication in overactive patients, immediate loss of consciousness, with amnesia for the whole treatment, and a low incidence of complications, with few fractures and systemic reactions. The apparatus used in this country by Myerson has been described by Feldman and Davis.[72] The current source is a 60-cycle alternating current with a controlled range of voltage from 50 to 130 and a milliamperage from 50 to 750. Metal electrodes, with electrode paste, are attached to both temporal regions with a head band. The average patient required 70 volts at 350 to 500 milliamperes for 0.1 second to produce a convulsion.

Meggendorfer[73] treated 40 patients with schizophrenia, with very good results in 10 and temporary improvement in 10 more; the others were not benefited. All eight patients with manic-depressive psychosis were improved. Gonda[74] reported 58 per cent remissions in 29 patients with schizophrenia, and 87 per cent recovery in 8 patients with manic-depressive psychosis. Myerson[75] states that the psychoses of choice for treatment are the marked continuous depressions, and that the treatments may be given in the home or office. In schizophrenia, this method has the same general value as the use of Metrazol, and the indications for treatment are the same. No neurologic sequelae have been observed, but Gonda reported the fracture of both humeri in 1 case. Precautions must be taken against fractures, as with any convulsive therapy, and the equipment must be tested before each treatment. The changes in the human central nervous system are unknown, since there have been no deaths. Bini,[70] however, has reported changes in animals that were in some cases irreversible, although not described in detail. Other observers have stated that the changes in animals were not so severe as those found after insulin hypoglycemia.[71]

Infantile cerebral palsy occurs in about 7 births

per 100,000 population a year and presents a problem in treatment. Spastic, athetoid and ataxic cases should be differentiated, since the treatment varies accordingly. Institutions where muscle re-education and schooling can be carried out together are most effective. Home therapy is very satisfactory in some cases but must be individualized, and the nature of the disorder and the personality must be understood by the family. The program of treatment consists essentially in special relaxation and co-ordination exercises, which must be persisted in daily and combined with occupation to form new habit patterns. Several recent publications[76–82] have been found helpful by parents and workers in the field.

The exact value of electrical stimulation in peripheral-nerve lesions to lessen atrophy and promote return of function is still problematical. In experiments on sciatic section in monkeys[83] and dogs,[84] it has been found that the degree of regeneration of peripheral nerve fibers and the atrophy and degeneration of denervated muscles are uninfluenced by physical therapeutic methods, including electrical stimulation. Massage and passive movement seemed most beneficial in securing maximum return of function, provided there was adequate splinting. Fischer,[85] however, showed in rat experiments that twelve to twenty minutes of intensive electrical stimulation produced a training effect in denervated muscles that lessened the degree of atrophy. Clinical experience has led most observers to consider electrical treatments beneficial in facial-nerve paralysis, but large series of cases and controls will be necessary to obtain more objective evidence of the value of electrical stimulation.[86]

Dermatology

The dermatologist is particularly interested in ultraviolet irradiation, because so many skin diseases have been given this therapy with both beneficial and deleterious results.[87] Blum[88] has recently made an outstanding contribution to the literature on the subject of light in relation to medicine. He carefully defined photodynamic action and described methods of investigation that should lead to more accurate studies in the future. Diseases due to light are considered in a separate section. It is essential that clinicians be aware of these conditions, since ultraviolet irradiation may be extremely dangerous, as in lupus erythematosus. Of late, the photosensitizing action of the sulfonamides has been frequently reported.[89–91] Sulfanilamide in high concentration sensitizes human skin to irradiation below 3200 Angstrom units, producing severe sunburn. This is not photodynamic action, and porphyrins are not involved, but it probably represents an increased sunburn reaction. Patients receiving sulfonamides should therefore not be exposed to sunshine or ultraviolet lamps. This photosensitizing action of sulfanilamide has been used in the treatment of psoriasis to enhance the effect of ultraviolet irradiation, but is not recommended routinely because of the potential dangers.[92]

Several reports have appeared on the successful treatment of fungous infection of the hands and feet by copper iontophoresis. Hoechstetter[93] treated 18 cases, with healing in 16, giving five to nine treatments weekly, or twice a week when the infection was severe. Equally good results were obtained by Haggard, Strauss and Greenberg.[94]

Otolaryngology

Local heat in various forms gives some symptomatic relief from upper respiratory-tract infections. A simple infrared lamp is quite effective for this purpose. Short-wave diathermy has been used extensively for sinusitis, but does not effect a cure.[95] Better results are obtained in acute cases than in chronic ones, but postoperative results are disappointing. In the treatment of gingivitis, paradentosis and postextraction pain, short-wave diathermy gave relief in all 64 of Kobak's[96] patients. Ion transfer (iontophoresis), using zinc salts for hay fever, vasomotor rhinitis and chronic otorrhea, has not brought the cures first hoped for. Evidence for proper selection of cases awaits further investigation.[97]

Fever Therapy

Artificial fever continues to have a role in the treatment of syphilis, gonorrhea and chorea. The methods of production and effects have recently been summarized.[98] Results and hazards are about equal with inductive heating, hot moist air and hot sprays of water.

In general paresis, results have been slightly better with artificial fever than with malaria.[99] Solomon, Kopp and Rose[100] have tried the effect of increasing the temperature swing during a six-hour period by first reducing the temperature to 90°F. under Pentothal anesthesia with ice packs, and then inducing a fever of 104 to 105°F. They have as yet made no conclusions of its value.

The sulfonamides have subordinated fever therapy in the treatment of gonorrhea, but the percentage of resistant cases has been reported from 10 to 20.[101–103] These results are about the same as those of fever alone. The combined use of sulfanilamide and fever, however, has given nearly 100 per cent cures.[104–107] Medication for eighteen to forty-eight hours prior to fever therapy has been recommended. A single ten-hour fever at

106 to 107°F. has then been sufficient to obtain negative cultures.

Good results of treatment of chorea by artificial fever continue to appear.[108–110] Short sessions (two and a half to three hours) at 104 to 105°F. are repeated daily or on alternate days for a total of about twenty to thirty hours of fever as the usual routine. Bennett and Hoekstra[110] had excellent responses in all 17 of their cases; Elkins and Krusen[109] reported only a single failure in 20 cases adequately treated.

Hyde[111] has tried short (one hour or less) and mild (up to 103°F.) fevers in allergic diseases, with interesting results. Thirty-two patients were treated, with improvement in 75 per cent of the asthma cases and 81 per cent of the hay-fever cases.

An increase in temperature from 98 to 103°F. has been shown to increase the bacteriostatic power of sulfanilamide about one hundred times.[112] With this in mind, combined fever and chemotherapy has been tried in subacute bacterial endocarditis. Bierman and Baehr[113] treated 16 cases, with 2 cures, two years and ten months, respectively, after treatment. Krusen and Bennett[114] reported 6 unsuccessfully treated patients. A review of the literature[115] revealed that among 200 cases due to *Streptococcus viridans* and nonhemolytic streptococcus, 6 per cent of the patients recovered after administration of sulfonamides. Of 43 patients treated with heparin and chemotherapy, there was 12 per cent recovery. Among 24 patients treated by chemotherapy and artificial fever, 4 recovered. Of 21 patients treated by chemotherapy and typhoid-paratyphoid vaccine, 5 recovered, an incidence of 25 per cent. These results are encouraging, but not conclusive, because of the small number of cases and short follow-up periods in some.

Physiology and Biophysics

Numerous reports have appeared of research on the physiologic effects of heat, light, electricity and other physical agents. Bisgard and Nye[116] found that gastrointestinal motor activity, as indicated by kymographic recordings, was inhibited by the application of heat to the abdominal wall and by ice water taken by mouth. Stimulation resulted from application of ice externally and from ingestion of hot water by mouth. Another study on the effects of hot and cold applications revealed that the nasopharyngeal irrigations at temperatures above 41°F. and below 113°F. caused no marked variation in cervical lymph flow. At temperatures of 41°F. or below and at 113°F. or above, the lymph flow increased during the application or for longer periods if the capillaries were damaged.[117] Gammon and Starr[118] investigated the relief of pain by counterirritants and found that the intermittent application of heat and cold was much more beneficial than the use of either agent alone. Changes in circulation did not explain the relief of pain; in animal experiments, action potentials from sensory nerves were altered, suggesting a neural mechanism.

The cardiovascular effects of heat have been reviewed by Bazett,[119, 120] and the present knowledge of the effect of heat, massage and exercise on lymphatic circulation has been authoritatively discussed by Drinker.[121] Diathermy has been shown to have no athermic effects on blood pressure[122] or intestinal activity[123] in animal experiments. It was useful in raising the cutaneous temperature of feet reflexly by application to the trunk.[124]

In studying reactive hyperemia, Abramson and Ferris[125] found that local heat to the hand was a more potent stimulus than tissue anoxia from ten-minute circulatory occlusion, whereas the reverse was true in the forearm and leg. Five-minute circulatory arrest resulted in relatively complete vasodilatation, according to Eichna and Wilkins,[126] and the reactive hyperemia was only slightly increased by local heat at 104°F. or prolonged ischemia (ten to fifteen minutes).

Iontophoresis may cause small but slowly healing burns in spite of the usual precautions. Molitor and Fernandez[127] made a careful experimental study of the problem and recommended definite current values for electrodes of various sizes. They have expressed this relation of safe current to electrode size in the form of a graph, which should be of value in improving technic and in preventing burns.

Other studies on technic and dosage have been reported in relation to short-wave diathermy. These should be of interest to technicians and have been abstracted in detail by Kovács.[128]

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 27401

PRESENTATION OF CASE

A forty-seven-year-old Greek laborer was admitted to the hospital because of a recurrent pain in the chest, accompanied by shortness of breath and general poor health of nine days' duration.

He had been followed in the Out Patient Department for ten years because of recurring attacks of pain, which radiated over the entire anterior part of the chest, the scapular area and both arms. Since the blood Hinton and Wassermann reactions were strongly positive, antisyphilitic therapy was given on several occasions. In all, about two hundred injections of either arsenic or bismuth (mainly the latter) had been given, followed by Mapharsen in small doses. Eight months before admission, the blood Hinton reaction remained positive, although the Wassermann reaction was negative. From the time that the patient was first studied, his heart and great vessels had been enlarged, and he had had an auricular flutter with variable block, usually approaching 4:1. A to-and-fro murmur was always present, loudest in the third left intercostal space. The blood pressure ranged around 140 systolic, 70 diastolic, until the last year, when the diastolic pressure failed, with resultant increase in pulse pressure.

At first, x-ray examination showed dilatation and increased pulsation of the aorta, accompanied by considerable dilatation and pulsation of the pulmonary conus and the hilar shadows. Subsequent roentgenograms demonstrated interesting changes in that the shadow of the heart margin became markedly irregular just above the apex, while the pulmonary conus and ascending aorta appeared to be involved in a double mass. The pulsations of this mass were less vigorous than those of the apex of the heart. The trachea was displaced to the right, and the left main bronchus was unusually small and irregular. On still another roentgenologic examination it was thought that definite, multiple, pulsating saccular dilatations extended both anteriorly and posteriorly from the root of the aorta. There was an apparent slight erosion of the left fourth rib near the costochondral junction.

Until entry into the hospital, the patient had remained free from pulmonary congestion, clubbing of the fingers, ankle edema and cough. He continued at heavy labor with pick and shovel until the last two weeks before entry, although he was cautioned repeatedly against doing so. Two years before the time of his final episode of illness, he had been digitalized, and had continued on this drug intermittently.

Nine days before hospitalization, there was a vague onset of ill health. A day or so later, the old chest pain returned. The patient had been free of this for a long time previously. Simultaneously, he began to experience shortness of breath, and difficulty in getting his breath. On the fourth day before entry, he began to redigitalize himself (having been without digitalis for at least two weeks). The following day he took to his bed. Because of exacerbation of the chest pain, his physician prescribed morphine and nitroglycerin, but neither drug afforded relief.

The past history was significant in that the patient had been a marathon runner while in his twenties. There was a definite history of a penile sore. His family history was irrelevant.

On physical examination, the patient was found sitting upright in bed in some respiratory distress but free from cyanosis. The heart was markedly enlarged, with an increase in the supracardiac dullness. A slight systolic thrill was felt over the third left interspace with a corresponding loud systolic murmur and softer diastolic murmur best heard in this area. There were marked pulsations in the veins of the neck. A loud friction rub was present over the left anterior chest above the heart, together with many coarse, crackling rales, which extended along the sternal border. Some dullness, without rales, was manifested at each pulmonary base. The blood pressure was 140 systolic, 40 diastolic. The pulse was of Corrigan type, with a rate of 84. The rhythm was totally regular at times, and irregular at others. The liver and spleen were not enlarged, and there was no peripheral edema.

The rectal temperature was 102°F., and the respirations 36.

Examination of the blood showed a white-cell count of 13,900 with 90 per cent polymorphonu clears, and a red-cell count of 5,150,000 with 90 per cent hemoglobin. The blood Wassermann and Hinton reactions were positive.

An electrocardiogram showed depression of the ST takeoffs in Leads 1 and 4, and their elevation in Lead 3. The ST segment sagged in all leads, and T was deeply inverted in the first three leads. No axis deviation was present.

An x-ray film showed the heart shadow to have enlarged about 2 cm. since examination in the clinic a month previously. The shadow in the

area of the great vessels was also larger. The left lower lobe of the lung appeared collapsed, with increased aeration of the left upper lobe.

During the night, the patient rested fairly well, although the pain in his chest was only partly relieved by sedatives. The respiratory rate slowed.

The following afternoon, he was found in coma, from which he could not be roused. He was cyanotic, breathing deeply and irregularly about four times per minute. The heart sounds were faint, at 20 per minute. Neither pulse nor blood pressure could be obtained. The patient was given adrenalin and coramine, and rallied slightly. He became restless, and complained of pain in his abdomen. Administration of oxygen improved his color, but both heart and lungs failed within fifteen minutes.

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Differential Diagnosis

Dr. Howard B. Sprague: It seems as if the fundamental situation here must be related to a syphilitic infection. The patient had received a good deal of antisyphilitic therapy. On the other hand, his cardiovascular examination was apparently abnormal by the time he started this treatment. The diagnosis of the anatomic changes appears to rest largely on the x-ray examination. It is true that all this time there was a to-and-fro murmur, loudest in the third intercostal space, which may have been due to true syphilitic involvement of the aortic valve, with aortic regurgitation. One must accept this description, although the murmurs were somewhat more obvious to the left of the sternum. It is fair to say that this does represent aortic regurgitation because we have the statement that he had a Corrigan pulse, although his diastolic pressure was maintained at 70 until the last year before entry, when it finally dropped and the pulse pressure increased. It sounds, then, as if there had not been a high degree of aortic regurgitation. The main pathologic lesion lay beyond the aortic valve.

I should like to have Dr. Schatzki tell me about this x-ray examination, because at one time we are told that the shadow of the heart's margin became markedly irregular, and later on it was thought that definite, multiple, pulsating saccular dilatations were present. I wonder if these saccular dilatations of the aorta made the margin of the heart shadow irregular.

Dr. Richard Schatzki: When one reads the story and hears about the aortic signs and about syphilis, everything seems to be simple. Aortic aneurysm is the first diagnosis. The films, however, are disturbing because they look different from those seen in the usual aortic aneurysms. The lesion is fairly well described in these reports. The striking thing is that this mass lies anteriorly, which means, if it is connected with the aorta, that it comes from the ascending aorta. Usually, if there is an aneurysm of the ascending aorta, it is on the right side of the heart in the anteroposterior films. The abnormality in the anteroposterior film in this case is almost exclusively on the left side. What lies in this area on the left side anteriorly? If it has anything to do with the vascular system, there are two possibilities. One is the pulmonary artery, and the other is the aorta, which is quite close to the pulmonary artery. Which of the two is it? Films taken later show that this mass had grown, but it remained in the same position anteriorly.

Dr. Paul D. White: What is the interval between those two films?

Dr. Schatzki: Two years. This is a film that was taken on the last day. It is important because it shows the mass and, within the shadow of the mass, a structure that has the appearance of the left hilus or pulmonary artery before it enters the hilus. It would be unusual for the pulmonary artery to be visible if the mass arose from the pulmonary artery.

Dr. Sprague: You do not say that this is the pulmonary artery.

Dr. Schatzki: Not necessarily; but I think it is. I can speak freely because I do not know this case. I am in the same predicament as you. I think what we see within the shadow of the mass is the pulmonary artery or pulmonary conus. The fact that it projects out so far does not necessarily mean that it was enlarged. It may have been pushed over. These films were apparently taken after the last episode. They are not comparable with the first films, because they are taken in a horizontal position. The heart shadow has markedly increased in size, and the mass has still further increased in size even considering the magnification; the aortic shadow seems a little larger. This man had an enlarged heart in the region of the left ventricle and a mass apparently connected with the large vessels, which, so far as its position is concerned, could have arisen from the pulmonary artery, but from these films alone I think it is more likely that it arose from the left border of the ascending aorta.

Dr. Sprague: Dr. Schatzki and I agree, anyway. I am not going to be led off the trail by the fascination of syphilitic involvement of the pulmonary artery. We know that when we have thought that sort of thing was present we have been fooled. We are, I think, on safer ground if we say that this was an unusual aneurysm, which arose just above the aortic valve, with a certain amount of dilatation of the root of the

aorta and with some aortic regurgitation, and that the systolic murmur was probably produced in this aneurysmal sac. It undoubtedly displaced the pulmonary artery, and it produced a constriction of a bronchus, resulting in collapse of the left lower lung, but I do not see enough in the picture pointing to trouble in the pulmonary circuit to make me consider this an aneurysm of the pulmonary artery, or perforation from the aorta into the pulmonary artery. There was a growth of this aneurysmal sac, with pain from actual erosion of the chest wall.

Let us discuss a few points in detail. It is unusual for a patient with a syphilitic heart to have auricular flutter. Of the patients autopsied here who have been studied electrocardiographically, I can recall only one with auricular fibrillation. One notes that there were marked pulsations in the veins of the neck, which I assume must have been caused by tricuspid regurgitation. There is no note that these resembled rapid flutter waves. The electrocardiogram shows inversion of the T waves in all three leads, with a depression of the ST takeoffs in Leads 1 and 4 and an elevation in Lead 3, all of which are suggestive, I should think, of some left ventricular strain, even though there is no increase in QRS with a downward direction in Lead 3, which is characteristic of left-axis deviation. That is not too unusual in syphilitic aortitis. About half the patients who have syphilitic aortitis with aortic regurgitation have normal electrical axes, as judged from the direction of QRS. There have been occasional cases of bundle-branch block, both right and left, and an occasional case of right-axis deviation, but in that event there has always been a complication. In this particular case, there might be, I suppose, the added strain on the right side of the heart, if there were an aortic aneurysmal sac distorting or compressing the pulmonary artery, which might lead to some balancing effect in the electrocardiogram.

The final episode is probably explained by the story of what appears to be an enlarging aneurysmal sac, with pain, friction rub and some leukocytosis. It is fair to say that the cause of death was a rupture of the aneurysm. If the aneurysm ruptured into any place where free bleeding could result, whether into a pleural cavity or into the esophagus, trachea or bronchus, I think that the patient would have died sooner than he did. A final x-ray report mentions a marked increase in the heart shadow, and there is a note that the heart sounds were faint, with marked bradycardia. Neither pulse nor blood pressure could be obtained. There is the syndrome of cardiac tamponade. The position of the aneurysm suggests that it ruptured into the pericardium. I am going to assume that that is what happened. In view of the position of this sac at the very base of the aorta, one could guess that there would be some obstruction in the region of one or the other coronary ostia. It does not quite seem as if he had an acute coronary closure as the final episode, although I imagine that that might be possible, or that perforation might in some way have resulted in further compression of the coronary artery. I therefore believe that this was a syphilitic aortitis, with aneurysm of the first portion, presenting to the left and anteriorly, and with final rupture into the pericardium.

DR. WHITE: Just to corroborate Dr. Sprague's opinion about the rarity of pulmonary-artery syphilis and pulmonary aneurysm, I want to say that the most important case that we followed for some years, believing that this was the diagnosis, proved at autopsy to have an aortic aneurysm presenting in the region of the pulmonary artery. That is also true of several other cases on record. Thus, our experience with pulmonary-artery aneurysm has thus far always been disappointing. There is, I think, some suggestion, as Dr. Sprague mentioned, that the pulmonary artery or its branches were compressed by an aortic aneurysm, thereby increasing the size of the main trunk and major branches of the pulmonary artery, along with a shift in position to the left. An increased pulmonary-artery pressure might have overburdened the right ventricle, accounting for the increased pulsation of the neck veins. Another explanation of increased fullness of the neck veins would be mediastinal compression by the aneurysm; that is not so likely, however, because there would then have been engorgement, with little or no venous pulsation. Rupture of the aorta into the pulmonary artery would have produced a continuous murmur, which was not present in this case, and rupture into the superior vena cava would have caused a marked increase in pulsations in the neck veins and also a continuous murmur, which was not present. One case of rupture of the aorta into the pulmonary artery, which was correctly diagnosed before death, had a continuous murmur that made the diagnosis possible. Also, rupture of the aorta into the superior vena cava would have been attended by overwhelming symptoms and a much shorter life than this patient had after the episode. I agree that death in this case was probably due to cardiac tamponade. Hemorrhage into the pericardium is often rather slow and allows a good many hours before death occurs.

Clinical Diagnoses

Pulmonary embolism.
Syphilis.
Syphilitic aortitis.
Aortic aneurysm?
Aneurysm of pulmonary artery?
Dissecting aneurysm?

Dr. Sprague's Diagnoses

Syphilitic aortitis, with aortic regurgitation.
Aneurysm of ascending aorta, with final rupture into the pericardium.

Anatomical Diagnoses

Syphilitic aneurysm of the aorta, with rupture into the pericardium and pulmonary artery.
Hemopericardium.
Syphilitic aortitis.
Pulmonary atelectasis.
Hydrothorax, left.

Pathological Discussion

Dr. Benjamin Castleman: This man did have a syphilitic aneurysm of the ascending aorta, which presented anteriorly, as suggested by Dr. Sprague and Dr. Schatzki. At the time of autopsy it measured 10 by 7 by 4 cm., quite a sizable aneurysm, filled, as usual, with a laminated thrombus. It covered most of the left auricle and presented more anteriorly than any we had seen before. It had ruptured from its posterior aspect into the pericardial sac just below the reflection of the pericardium, and there was 1000 cc. of fresh blood in the cavity. The aneurysm was apparently adherent to the pulmonary artery before rupture, because it also ruptured terminally into the pulmonary artery. The left lower lobe and lower portion of the upper lobe were collapsed, apparently because of pressure on the bronchus by the aneurysm, and there was 1000 cc. of straw-colored fluid in the left pleural cavity. The syphilitic aortitis apparently had not extended down to the aortic valve itself, since there was no separation of the cusps at their commissures, and nothing that would indicate that the patient had aortic regurgitation. The coronary mouths were not constricted. The mouth of the aneurysm was located 1.5 cm. above the valve, and the tear into the pulmonary artery had occurred 3.5 cm. above the pulmonary valve. The heart was not much enlarged.

Dr. Sprague: Was there no obvious increase in the size of the right ventricle?

Dr. Castleman: No; the right wall measured 3 mm.

Dr. White: That leaves open the explanation of the diastolic murmur. I suppose it is possible that during life the aortic ring was dilated, allowing some regurgitation that was not apparent when the aorta collapsed after death. The aneurysm was very low in position, was it not?

Dr. Castleman: Yes.

Dr. White: It has been my conviction for some time, but this is not generally recognized so far as I know, that in a good many patients with syphilitic aortitis the aortic regurgitation is due, not to a structural change in the valve, but to a dilatation of the very first part of the aorta, involving also the aortic ring. This is much more evident during life than at autopsy.

Dr. Sprague: That is borne out by the autopsies here. I have been going over them. There has frequently been no involvement of the valve, even though the medical records continuously report signs of aortic regurgitation.

CASE 27402

Presentation of Case

A sixteen-year-old American schoolgirl entered the hospital complaining of a skin eruption.

Four months before entry, the patient noted on each cheek a patch of redness that was associated with burning. The lesions spread until they crossed the bridge of the nose and assumed a butterflylike outline. Three months later, the redness had spread to her forehead. Two weeks before entry, while taking a bath, she noticed that there were red spots on her feet, and on further examination some were found on her back. Four days before admission, she developed a temperature of 101°F., and a few days later noted red patches in her mouth. Her weight had dropped from 150 to 132 pounds. Otherwise, she had been quite well, except for some loss of energy.

The father, mother and two siblings were living and well. There was no history of tuberculosis.

The patient had eczema as a baby and the other usual childhood diseases, including measles, pertussis, scarlet fever, diphtheria, chicken pox and mumps. She had spontaneous nosebleeds about once a month. There was no history of chest pain, night sweats or cough.

Physical examination showed a well-nourished, cheerful girl not acutely ill. The face revealed a masklike involvement, which excepted the mouth, eyes and hair margin. The lesion was described as a dusky erythema of a peculiar purplish hue, yellowish red at the outer margins. It was dry throughout, with a tendency to a fine, grayish scaling. The ears were involved, but the scalp

was free. There were hemorrhagic crusts in the nose. The buccal mucous membrane was denuded in several areas, and the hard palate ulcerated. The lesions were all rather sharply limited. The face was slightly tender and warm. There were a few reddened areas on the back and over the sternum. The tips of the fingers were reddened and slightly swollen. The cervical and left inguinal lymph nodes were enlarged and moderately tender. The edge of the spleen was felt on deep inspiration.

The temperature was 99°F., the pulse 90, and the respirations 20.

Examination of the urine was negative. The blood showed a red-cell count of 4,180,000 with a hemoglobin of 85 per cent and a white-cell count of 5800 with 56 per cent polymorphonuclears. The blood Hinton reaction was negative. The bleeding time was 2 minutes, the clotting time 2 minutes.

During the first week, the patient improved. The white-cell count, which had fallen as low as 2600, rose to 4000. The edema of her face was less marked, and the erythema of the lesions had subsided. About one month after admission, she had a slight fall in her white-cell count that was consistent with new lesions and swelling of the face. She was given nucleotide therapy for about two weeks, without any improvement. She developed generalized choreiform movements, particularly involving the left side, which persisted until death. She had weakness of the left hand, with circular movements. The knee jerks were active. She received x-ray treatment for her skin lesions, with remarkable improvement. After the development of choreiform movements, it was learned that the patient had been treated by a physician one year before entry for nervousness because "she could not sit still a minute." She developed a soft systolic murmur, which was believed to be hemic in character, probably owing to her secondary anemia. The temperature ranged from 99 to 102°F., and shortly after the third month of her stay it rose to 104°F. and she complained of headache, without any local signs or symptoms. Then the temperature rose to 105°F., and she became quite prostrated and dull and complained of a "funny feeling" in her head. The skin was hot and moist, and the skin lesions became redder. She developed pain in the right ear, and examination showed slight edema along one edge of the drum. A pericardial friction rub was heard for the first time. She rapidly failed, became semistuporous, and died three and a half months after admission — seven and a half months after the onset of her illness.

Differential Diagnosis

Dr. Wallace Zeller: The presence of acute disseminated lupus erythematosus is occasionally unsuspected because of the absence of the skin lesions so frequently found in this disease. In the case under discussion, no such oversight could occur. The skin lesions as described were quite consistent with this diagnosis, and numerous other features presented by this patient were also compatible. The patient belonged to the age group and sex most commonly affected by this disease, and the fever, weight loss, mucous-membrane lesions, lymph-node enlargement, redness and swelling of the fingertips, edema of the face and enlargement of the spleen are all characteristic findings in this widespread constitutional malady. We do not know definitely how long this patient had been troubled with spontaneous nosebleeds, so that their significance cannot be accurately weighed. Since purpura or other hemorrhages were not noted during the course before death, one is not justified in assuming that the nosebleeds were due to thrombocytopenic purpura, which is sometimes associated with systemic lupus erythematosus. The leukopenia, which was consistently found, is, of course, quite characteristic. The soft systolic murmur was thought to be of hemic origin. The degree of anemia at the time this murmur appeared is not stated. Such murmurs, however, may be caused by the endocarditis that sometimes accompanies acute disseminated lupus erythematosus. Their presence in other patients has, in fact, often led to an erroneous diagnosis of rheumatic fever. The terminal occurrence of pericarditis in this patient is the only evidence of involvement of the serous membranes. Headache such as she complained of has been noted in systemic lupus erythematosus, usually without any other evidence of involvement of the nervous system. It is possible that otitis media developed a short time before death. It might have been associated with a hemolytic streptococcus infection, which is often a terminal event in these patients.

There is much in this case history, therefore, to support a diagnosis of acute disseminated lupus erythematosus. Three items deserve special comment, however, since I believe they are unusual in this disease: First, the urine was described as normal, and it is implied that the patient had at no time demonstrated urinary abnormalities. Clinical evidence of renal disease is almost a constant feature of acute disseminated lupus erythematosus. Although it has occasionally been shown that kidney involvement may occur without clinical manifestations in this disease, evidence of its absence here must be weighed seriously in con-

sidering the diagnosis. Secondly, the presence of choreiform movements is, so far as I know, unusual in acute disseminated lupus erythematosus. Convulsions do appear in some cases shortly before death, but I know of none in which chorea has been described. The type and degree of involvement of the nervous system in disseminated lupus is not well known anatomically. Since the disease does involve the nervous system, however, and since widespread damage to small vessels is commonly found, it is not improbable that choreiform movements might occur. They would admittedly be very unusual. Thirdly, it is stated that improvement in the skin lesions followed x-ray therapy. Exacerbations of systemic lupus erythematosus following exposure to ultraviolet rays are well known. Similar exacerbations following roentgen-ray treatment have also been observed. It would be very unusual for exposure to x-rays to be followed by remission rather than exacerbation of the rash. It is not improbable that roentgen therapy was given to this patient on the chance that she might have mycosis fungoides. The fact that her skin improved temporarily following treatment is in favor of such a diagnosis rather than systemic lupus erythematosus. It should be noted, however, that x-ray treatment was not continued and was not tried again when the patient had the terminal exacerbation with further skin involvement. Apparently, the former benefits of the x-ray treatment were not considered to be very striking, or some contraindication to its use was thought to be present.

It is evident, then, that these three unusual features are of only relative importance in diverting us from a diagnosis of systemic lupus erythematosus. The disease can occur without clinical evidence of renal involvement.

Are there other diseases that should be considered in the differential diagnosis? Certainly, Sydenham's chorea in association with rheumatic fever and carditis should be thought of, although the skin and mucous-membrane manifestations would be difficult to interpret on this basis. Since they played so large a part in the clinical picture, I believe this diagnosis is entirely inadequate. The same reasoning can be applied to a consideration of subacute bacterial endocarditis. Since no comment is made regarding blood cultures, I assume that they were negative.

The possibility of dermatomyositis should be considered whenever a diagnosis of systemic lupus erythematosus is under judgment. As Keil[1] has shown, these two diseases may be almost identical clinically. Difficulties in distinguishing between them are usually encountered in patients with induration of the subcutaneous tissues and muscles, however, and this has not been described in the patient under discussion. The absence of abnormalities of the urine would by itself tend to favor a diagnosis of dermatomyositis as opposed to disseminated lupus erythematosus. Evidence in favor of acute disseminated lupus erythematosus rather than dermatomyositis is more imposing and includes the following: absence of involvement of the eyelids; the occurrence of pericarditis; the apparent absence of any real myositis; the definite enlargement of the lymph nodes; and the occurrence of symptoms referable to the nervous system.

It appears therefore that the probable diagnosis in this case is systemic lupus erythematosus. Since the post-mortem findings in patients dying of this disease are often very few, it is worth while to enumerate those which may be found in this case. The skin lesions, the large lymph nodes and spleen, the pericarditis, possibly with effusion, and the vascular lesions of the central nervous system may well be described. It will be interesting to find if there is gross or microscopic evidence of renal disease, since no urinary abnormalities were noted during life. Endocarditis may or may not have been present. It is quite likely that otitis media was found at death, and there may have been bacterial evidence of terminal septicemia. As has been pointed out in previous conferences here, the final diagnosis must be made by considering the post-mortem findings along with the symptoms and signs present before death.

Clinical Diagnosis

Acute lupus erythematosus disseminatus.

Dr. Zeller's Diagnoses

Acute lupus erythematosus disseminatus.
Otitis media.
Terminal septicemia?

Anatomical Diagnoses

Lupus erythematosus disseminatus, acute.
Acute endocarditis, terminal (bacterial ?).
Focal necrosis of myocardium.
Infarcts of spleen, kidneys and brain.
Otitis media, right.
Mastoiditis, acute suppurative, right.
Meningitis, acute.
Bronchopneumonia, early.
Pulmonary atelectasis.
(Chorea.)
Peritonitis, chemical, from perforation of stomach (probably post mortem).

Pathological Discussion

Dr. Tracy B. Mallory: As Dr. Zeller has pointed out, the anatomic findings in cases of acute disseminated lupus are often disappointingly few and are at best extremely inconclusive. On the other hand, when the clinical and the anatomic findings are added together, a symptom complex can be built up that is reasonably characteristic. I know of no anatomic finding that is conclusive. Even the skin rash may be very transitory, and no trace of it may be found at the time of death. In this case, it was still present in marked form; otherwise we should have been quite at a loss to make an anatomic diagnosis. A relatively frequent finding in these patients is a polyserositis, but in this case there was no inflammation of the pericardium or pleura and a localized patch of fibrin about the tip of the appendix was all that suggested peritoneal involvement. Enlargement of the spleen and lymph nodes, which is relatively constant, was found in this case. On histologic examination, acute necrosis of the germinal centers was apparent, a very characteristic point in the more acute cases. The heart was normal in size but did show an acute endocarditis of the mitral and aortic valves. From this source, emboli had developed and had produced small infarcts in the spleen and kidneys. The kidneys, furthermore, showed what is called focal glomerular nephritis. By this term we mean findings in occasional glomerular tufts or even in single loops of the tufts essentially like those of glomerular nephritis but with many entirely normal glomeruli. This is a frequent finding in disseminated lupus but is also characteristic of subacute bacterial endocarditis, so that in this case it does not serve as a differential point. There was, as was suspected, an acute otitis media and also a mastoiditis on the right. The cerebral symptoms were explained by the presence of a nasal meningitis and also of an early abscess in the right cerebellar lobe. No path of spread of the infection from the mastoid could be made out, so that we do not know whether the cerebellar abscess was secondary to the mastoid or was the result of a septic embolus. Unfortunately no cultures were made of either the endocardial vegetation or of the cerebellar abscess. The character of the endocardial lesion, however, leaves little doubt that the endocarditis was of the bacterial rather than the peculiar nonbacterial type often referred to as Libman–Sacks[2] endocarditis. In summary, it seems probable that we have been dealing with a case of acute disseminated lupus complicated, as these cases not infrequently are, by terminal sepsis, which in this case took the form of a subacute bacterial endocarditis.

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INTERN SUPPLY AND DEMAND

Hospital administrators expect to face continual new adjustments as the role of the hospital in medical care and medical education continues to change. The medical and surgical service rendered in the modern hospital has come to depend more and more on the activities of interns — usually, recent medical-school graduates who seek to gain practical experience and instruction to supplement their medical education. It gradually evolved that the hospitals and the interns each had a need for the facilities and services of the other, and ten years ago, there appeared to be an approximate balance between these mutual facilities and services in the United States. Some hospitals were then at pains to secure interns who were racially or culturally adapted to their patient clientele, but very few were confronted with a failing supply of interns. The primary concern of most hospitals was to secure and maintain the approval of their institutions as suitable places for young graduates to serve internships. They assumed, or perhaps were led to believe, that such approval would vouchsafe for them a continuing supply of interns. Ten years ago, it did.

Today, the problem has changed. Hospitals in general are less concerned with the qualifications of their interns, although they naturally want the best available, and many of them are even worried by the possibility of being unable to provide a fully staffed intern service. Refugees who cannot speak the English language fluently are in demand, and are actually securing histories from patients who cannot understand their questions. Today, there are in the United States one thousand internships for which there are no interns, and for which there are not likely to be interns for many years to come. The military exigencies facing the Nation are beginning to accentuate this situation, but one should not make the mistake of assuming that they have in any way brought this about: the cause is very plain and should be understood by all who are affected by or interested in the problem.

The annual number of medical-school graduates has changed very little during the past ten years; although there was a 20 per cent increase from 1930 to 1937, the number has again fallen off, so that the 1940 and 1941 figures are approximately identical with the average for the decade. Against this relatively fixed supply, there has been a steadily increasing demand for interns' services by the hospitals. Not only has the number of hospitals seeking to use and to train interns increased, but almost all that had had an intern staff have enlarged it. Hospitals have also increased the number of resident physicians in their employ, and in some institutions it has become a part of the hospital economy to give board and room to third-year and fourth-year medical students in return for their services during the evenings and week ends. Apparently, this last

type of hospital service is the least regulated, and can be utilized by hospitals that do not even apply for approval by the Council on Medical Education and Hospitals of the American Medical Association. This dislocation between supply and demand has been made even more stringent by the steady increase in the amount of laboratory work called for today, as compared with that requested ten years ago. Even when this is accompanied by a corresponding increase in laboratory personnel, the work of the interns is not simplified. Consequently, the hospitals of the United States are now engaged in a new type of competition — a competition for their rightful share of the limited supply of available interns.

Among the hospitals in certain metropolitan areas, regulatory procedures have been in operation for many years. So far as they concerned those areas and so far as they did not involve the fourth-year medical students in too many schools, these regulations worked well; but when one metropolitan area advanced the date of its examinations and stipulated that the students selected must immediately accept the appointments or refuse them forthwith, the next year another area was sure to advance the date again. Last year, it was generally agreed that appointments would not be made before a certain day in November, but many hospitals jumped the date when it seemed to be advantageous for them to do so. This year, at least one hospital has written to some of the medical schools asking for the names and addresses of all students in the upper third of the graduating classes — offering to pay the schools for the compilation of such a list — so that they might circularize these men and perhaps inveigle them in advance of other opportunities or commitments. The competition is becoming keen, and for the fourth-year medical student to be competed for rather than to be in competition is a refreshing situation. He who is being competed for, however, must be wary. He should develop sales-resistance, for he will inevitably be approached, proselyted and attracted by whatever devices hospitals can legitimately employ to secure their share of intern attention and service, and the institutions that talk the loudest should bear particular scrutiny.

Even if the medical schools could increase their output and thus satisfy the demands for intern service, — which at the present time they cannot do without lowering the standards of medical education, — this merely means that a serious oversupply of practicing physicians would eventually occur, since the very increases in the utilization of hospitals that have brought about the present disproportions have already diminished the practice of domiciliary medicine. Economic laws seem to point toward an arrangement whereby those hospitals that do not or cannot provide bona fide educational privileges to their interns would hire graduate interns as resident physicians, thus adding to the cost of medical care but, on the other hand, staking the fledgling physician to the price of an automobile and his first month's rent or the wherewithal with which to pay his debts when he eventually begins private practice.

JOURNAL OF CLINICAL ENDOCRINOLOGY

THE growing field of endocrinology has demanded another journal. In 1917, *Endocrinology* was begun and for many years this important publication, under the direction of the Association for the Study of Internal Secretions, has maintained a high standard of excellence. Papers, however, on the biochemical aspects of the endocrines from highly specialized laboratories have been printed in the same issue with clinical reports. The two aspects of the same problem are, of course, intimately related but may be easily separated without loss to either. Hence, a new journal, the *Journal of Clinical Endocrinology*, published under the same auspices, has been started; it reflects the advances in clinical studies and should prove to be of great use to practicing physicians. The senior publication will continue to report on laboratory investigations and will thus complement the new journal.

The *Journal of Clinical Endocrinology*, begun in January, 1941, with Milton Lee as managing editor and Charles C Thomas as publisher, is a

welcome addition to medical literature. With an editorial committee of distinguished contributors to the field of endocrinology, such as Fuller Albright, J. S. L. Browne, E. C. Hamblen, E. L. Sevringhaus and K. W. Thompson, the new journal starts with a sound foundation for a successful career.

MEDICAL EPONYM

Klebs-Loeffler Bacillus

The bacillus of diphtheria was discovered by Edwin Klebs (1834–1913), of Zürich, and reported at the Second Congress for Internal Medicine at Wiesbaden on April 19, 1883. The paper "Ueber Diphtherie [On Diphtheria]" is printed in the *Verhandlungen des Kongresses für innere Medizin* (**2:** 139–154, 1883). A portion of the translation follows:

> In form, these rods are of equal length, quite narrow and, on the whole, hardly as large as tubercle bacilli. A fair number are spore bearers, each rod always having two spores, one at each end.

Friedrich August Johann Loeffler (1852–1915), in a monograph entitled "Untersuchungen über die Bedeutung der Mikroorganismen für die Entstehung der Diphtherie beim Menschen, bei der Taube und beim Kalbe [Studies concerning the Significance of Micro-organisms in the Etiology of Diphtheria in the Human Being, in the Pigeon, and in the Calf]," was able to demonstrate by the application of Koch's postulates that the organism described by Klebs was indeed the etiologic factor in diphtheria. In this monograph, which was published in the *Mitteilungen aus dem kaiserlichen Gesundheitsamte* (**2:** 421–499, 1884), the author refers to previous investigations. A portion of the translation follows:

> We thus see that studies dealing with the significance of bacteria that occur in diphtheritic material have not as yet led to any satisfactory conclusions. It seemed imperative, therefore, to attack the solution of this important question with the help of the newest investigative methods, namely Koch's methods of culture on solid media, and thus to discover the significance of all these kinds of bacteria. It was necessary, first, to determine which varieties, by reason of their relation to the diseased tissues, seem to be chiefly concerned in the etiology of diphtheria, then to grow these in pure culture, and finally to undertake animal inoculation experiments with the pure cultures in as many species as possible.

There follows a detailed account of the successful investigation that enabled him to say in conclusion: "We thus have here two examples of perfect, infectious diphtheria of pure bacillary origin."

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

SOCIETY HEADQUARTERS

Owing to the shortage of fuel oil, the Boston Medical Library will be closed on Saturdays, commencing October 4 and continuing until late spring. This will necessitate the closing of the headquarters of the Society and the office of the Journal during the corresponding period. In an emergency, the officers of the Society and the managing editor and associate editors of the Journal will be available through their offices or homes.

SECTION OF OBSTETRICS AND GYNECOLOGY*

Fatal Fibrocaseous Tuberculosis and Femoral Thrombophlebitis Following Cesarean Section

A twenty-four-year-old primipara entered the hospital at term for an elective cesarean section four hours after the rupture of the membranes. The indication for the cesarean section was a small round pelvis.

The heart and lungs were reported to be normal. The patient was first seen when approximately sixteen weeks pregnant and was followed routinely and adequately.

On admission to the hospital, the temperature was 98°F., the pulse 120, and the respirations 20; when the patient returned from the operating room, the temperature was 101°F., the pulse 92, and the respirations 24. From then until the fortieth postoperative day, when she died, the patient ran a continuous fever,—a temperature of 98 to 103°F.,—respirations of 30 to 35, and a pulse of 110 to 120. At some time during the puerperium, a right femoral phlebitis developed. A medical consultant saw the patient and noted that there was evidence of a very rapidly developing miliary tuberculosis. The patient was transfused several times and treated conservatively.

Autopsy showed bilateral fibrocaseous tuberculosis, with extensive cavity formation, and right femoral thrombophlebitis.

Comment. It is very hard to understand how such an extensive tuberculous process could have existed during pregnancy without being discovered. The operation was performed solely because of pelvic disproportion, and it was not until the temperature rise called attention to the lungs

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section. Letters should be addressed to Dr. Raymond S. Titus, *Secretary*, 330 Dartmouth Street, Boston.

that the pulmonary process was recognized. It is perfectly possible, in fact probable, that an old tuberculous process was lighted up by the infection that followed the cesarean section. Hence, the cesarean section was probably the indirect cause of death. If the infection had not resulted, the tuberculous process would probably have remained not only quiescent but entirely unrecognized.

Tuberculosis is not a common complication of pregnancy. Patients with mild cases under hospital care may go through pregnancy without undue risk, and it is only in advanced cases that fatalities ensue.

DEATHS

CHOLERTON — Herbert Cholerton, M.D., of West Somerville, died September 25. He was in his sixty-ninth year.

Born in Manchester, England, Dr. Cholerton received his degree from Harvard Medical School in 1901. He was a fellow of the Massachusetts Medical Society and the American Medical Association.

He is survived by his widow, his mother, a sister, and a brother.

KELLEHER — Patrick F. Kelleher, M.D., of Cambridge, died September 25. He was in his seventy-fourth year.

Dr. Kelleher received his degree from Tufts College Medical School in 1896. He served on the staffs of St. Elizabeth's and Cambridge City hospitals, and was a fellow of the Massachusetts Medical Society and the American Medical Association.

His son and a sister survive him.

LANE — Edward B. Lane, M.D., of Milton, died September 17. He was in his eighty-second year.

Born in Melrose, he attended Boston University and graduated from Harvard University. He received his degree from Harvard Medical School in 1885 and served his internship at the McLean Hospital, after which he became assistant physician at the Northampton State Hospital. In 1895, he was appointed first superintendent of the Boston State Hospital, a position that he held until 1905, when he retired to private practice.

From 1898 to 1903, Dr. Lane was clinical instructor in mental diseases at the Harvard Medical School, resigning to become professor of mental diseases at the Tufts College Medical School; he retained this position for twenty-five years. He was formerly resident physician at the Adams Nervine in Jamaica Plain. He was a member of the Massachusetts Medical Society, the American Medical Association, the American Urological Association, the American Psychiatric Association and the Dorchester Medical Club.

His widow, three sons and a sister survive him.

MALLORY — Frank B. Mallory, M.D., of Brookline, died September 27. He was in his seventy-ninth year.

Dr. Mallory received his degree from Harvard Medical School in 1890. He was professor of pathology at the Harvard Medical School for many years and was head of the Pathological Laboratory, — later called the Mallory Institute of Pathology, — Boston City Hospital, from 1897 until his retirement in 1933. He was a fellow of the Massachusetts Medical Society and the American Medical Association and a member of the American Association of Pathologists and Bacteriologists, having served as its treasurer for many years. Dr. Mallory was the author of two standard pathological textbooks and many original articles, and for fifteen years was editor of the *American Journal of Pathology*.

He is survived by a sister, Miss M. Eleanor Mallory, of Avon Park, Florida, and by two sons, Dr. Tracy B. Mallory and Dr. G. Kenneth Mallory, both of Brookline.

TOBEY — George L. Tobey, Sr., M.D., of Medonak, Maine, died September 18. He was in his eighty-ninth year.

Dr. Tobey formerly served on the staff of the Clinton Hospital, and had been district medical examiner. He was a former member of the Massachusetts Medical Society, and was a member of the Maine Medical Association and the American Medical Association.

His sons, Dr. George L. Tobey, Jr., Dr. Harold G. Tobey, and Guy D. Tobey, survive him.

CORRESPONDENCE

INFORMATION CONCERNING THE ARMED FORCES IN PHYSICIANS' WAITING ROOMS

To the Editor: During the past few weeks, several medical men in New England have informed this headquarters that they would be pleased to take a more active part in the national defense effort. In particular, several physicians have signified their desire to secure accurate information about current opportunities in the United States Army for young men who were their patients or relatives and friends of patients.

In this connection, it has also been suggested that many professional offices would lend themselves most fittingly to the display and possible distribution of reliable information about the armed services in the form of official publications, which might be included with the other standard reading matter of a physician's waiting room.

May I take this opportunity to invite any physician in New England who wishes to join others in this vital phase of national defense to notify the Commanding General, First Corps Area, Army Base, Boston, Attention CARO? Appropriate material will promptly be furnished.

The fine spirit that your profession has already displayed in innumerable ways during the current emergency makes me sure that all its members who find it possible will co-operate in this matter.

F. B. Wilby
Brigadier General, United States Army
First Corps Area
Army Base, Boston

PREMARITAL MEDICAL EXAMINATIONS

To the Editor: I am much interested in House Bill No. 460 dealing with premarital physical examinations, a transcript of which appeared in the August 28 issue of the *Journal*.

Section 20B states, "If such physician, in making such examination, discovers evidence of any infectious disease declared by the state department of public health to be dangerous to the public health, he shall inform both parties of the nature of such infectious disease and of the pos-

sibilities of transmitting the same to his or her marital partner or to their children. Such examination shall include a standard serological test for syphilis. . . ." If either applicant is suffering from one of the more obvious reportable diseases, like smallpox or diphtheria, the physician's problem is relatively simple. But it is quite evident from the demands for a serologic test for syphilis that the intention of the bill is likewise to exclude genitoinfectious disease in both applicants.

Shall the physician rest content with a cursory inspection of the antenuptial phallus, or shall he also massage the prospective bridegroom's prostate to secure microscopic evidence of purity of soul and sanctity of habits? And if so, should he be content with aught but cultural evidence of noninfectiousness? And what of the blushing bride? To rest content here with a negative Hinton test and a throat and skin free of obvious infection is hardly to carry out the purpose of this legislation. Are we to certify that the lady is free of "infectious disease . . . dangerous to the public health" when we have not the remotest idea of the existence or character of vaginal discharge? And shall we be satisfied with merely a pudendal smear, or must we go into the problem more deeply? Here we are faced with a perplexing variety of social taboos and sentimental prejudices.

To imply ridicule of this estimable piece of legislation is farthest from my thoughts; but on the shoulders of every practicing physician has descended a very real responsibility to the State as well as to the patient. How thorough an examination should be performed in order to carry out the spirit and the letter of this law and to enable the physician to assert without fear of serious contradiction that neither party harbors a reportable contagious disease? Unless frank discussion of this problem produces some unanimity of opinion and of practice the whole thing can become a farce that merely creates a sense of false security in the minds of the affianced.

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POST-PARTUM EMBOLISM

To the Editor: In the August 28 issue of the *Journal*, under the Section of Obstetrics and Gynecology of the Massachusetts Medical Society, a case of post-partum phlebitis and fatal pulmonary embolism was presented, and the assertion was made that death might have been averted had the patient been kept in bed for a longer period. The fever was assumed to have been due to pelvic thrombophlebitis, although no evidence other than fever is given to support this diagnosis. On the other hand, the presence of tenderness in the leg, together with fever, should have been regarded as presumptive evidence of thrombophlebitis of the deep veins in the lower leg.

The assumptions made in that report deserve revision in view of the recent evidence concerning the etiology of pulmonary embolism: to wit, that the vast majority of emboli arise from thrombophlebitis of the deep veins of the lower leg; that the treatment of such thrombophlebitis by prolonged rest in bed is not an effective safeguard against the occurrence of embolism; that the existence of an unexplained fever and tenderness in the lower extremity makes it incumbent on the physician to make or dismiss the diagnosis of thrombophlebitis on the basis of x-ray visualization of the deep veins by Bauer's technic; that once the diagnosis is made the only certain prophylaxis against embolism is immediate ligation of the femoral vein proximal to the clot, or heparinization, which is less desirable for reasons already pointed out by various writers.

In the case referred to, it is clear from existing data that keeping the patient in bed for a longer period would not have guaranteed the avoidance of embolism, whereas ligation performed in time and in the right circumstances would. Gynecologists and obstetricians should revise their assumption that the site of origin of emboli in their cases is from the pelvic veins. This belief continues to render them helpless to avoid the calamity of embolism. The facts warrant an optimistic view in regard to an effective prophylactic technic against embolism. Careful observation of the lower extremities in post-partum and post-operative patients should lead to a sharp reduction in the incidence of fatal pulmonary embolism.

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DEFENSE COUNCILS

To the Editor: According to a joint statement issued on September 4 by the director of the Office of Civilian Defense, F. H. LaGuardia, and the chairman of the American National Red Cross, Norman H. Davis, state and local defense councils are the official agencies responsible for the co-ordination of all available resources that may be required for civilian protection in the event of belligerent action. Defense councils should therefore acquaint themselves with the resources of the local Red Cross chapters in providing food, clothing, shelter, nursing care, transportation and other basic necessities and should integrate them into the comprehensive local program. Duplication of trained and experienced personnel and of available supplies of the Red Cross should be avoided except where supplementation is essential to meet the anticipated needs of the community.

GEORGE BAEHR, M.D.
Chief Medical Officer

Office of Civilian Defense
Washington, D. C.

BOOK REVIEWS

Scabies—Civil and Military: Its prevalence, prevention and treatment. By Reuben Friedman, M.D. 8°, cloth, 288 pp. New York: Froben Press, 1941. $3.00.

The subject of scabies is thoroughly reviewed in this book, particularly in its historical and military aspects. Over two hundred pages are devoted to the importance of scabies in relation to war, but there are also chapters on scabies in Colonial America and similar historical data. The material, in general, is badly presented, and the book is difficult to read. Considerable semi-irrelevant or badly digested facts are included. Moreover, the format is unattractive, and in many places there is evidence of lack of care in printing. The book, although it contains some material of interest, is not one that can be enthusiastically recommended.

Textbook of Medicine. By various authors. Edited by J. J. Conybeare, M.C., D.M. (Oxon.), F.R.C.P. Fifth edition. 8°, cloth, 1131 pp., with 24 illustrations, and 31 x-ray plates. Baltimore: Williams and Wilkins Company, 1940. $7.50.

The fifth edition of this book, which was first published in 1929, is an indication of its worth to the medical

profession. Made up of contributions by various authors, but edited by Dr. Conybeare, physician to Guy's Hospital in London, this volume has long maintained an important place in the world of medical textbooks. This edition contains a great deal of new material, for many sections have been partially rewritten and some completely revised. Articles on sulfonamide therapy, blood transfusion, herniation of the nucleus pulposus and similar modern additions to medical knowledge are included. It has not been possible, however, to keep the book strictly up-to-date, largely because of the emergency created by the war in England. The authors are apparently not acquainted with many recent additions to medical literature, although the volume maintains its position as an important textbook.

Medical Diseases of War. By Sir Arthur Hurst, D.M. (Oxon.), F.R.C.P., with the co-operation of H. W. Barber, M.D. (Cantab.), F.R.C.P., F. A. Knott, M.D. (Lond.), M.R.C.P., and T. A. Ross, M.D. (Edin.), F.R.C.P. 8°, cloth, 327 pp., with 37 illustrations and 4 plates. Baltimore: Williams and Wilkins Company, 1940. $5.50.

This book, a new edition of one first published in 1916 and republished in 1918, will be found especially helpful to neurologists and to all interested in the neuroses of war. It is based largely on the author's wide experience in World War I as neurologist to Guy's Hospital, officer commanding Seale-Hayne Hospital for Functional Nervous Disorders and consulting physician to the Salonika Army, but under conditions so far experienced it may not be so applicable in World War II.

When one realizes that almost 6 per cent of the 341,025 discharges of soldiers from the British Army from 1914 to the end of April, 1918, were due to war neuroses, the value of recognizing and treating these cases correctly is evident. Hurst's remarks in Chapter I, "Predisposing Causes of War Neuroses," will repay careful reading. Other chapters are entitled "Hysterical Symptoms in Soldiers," "Hysterical Contractures," "Hysterical Postures and Gaits," "Hysterical Tremors" and "Hysterical Blindness"; they contain numerous brief case records, which are instructive. Many results and cures are almost miraculous.

Of especial interest is Chapter XVI by T. A. Ross, entitled "Anxiety Neuroses of War." Here, in particular, psychotherapy, with explanation to the patient, persuasion and re-education, has worked wonders. The chapter, "Soldier's Heart," is very good; in great measure, this is based on Sir Thomas Lewis's book, published in 1918. The section by H. W. Barber, "Skin Disease in War," is of special value. Medical officers of the rapidly expanding Army of the United States, an army composed of men who are very liable to be infected in crowded barracks, should study this section with care.

The title of this volume is somewhat misleading, since almost half of the book is given over to the neuroses of World War I, and the other half concerns almost entirely a number of medical diseases, such as trench fever, typhoid and paratyphoid fevers, bacillary dysentery, epidemic jaundice and tetanus, all of which promise not to be important in World War II, because conditions are so different from those in 1914–18. One expects to have epidemics and perhaps pestilence, but more probably among the civilian population than among the military forces.

Of great value are the references to the published literature, at the end of each chapter. An index also adds to the general usefulness of the volume. This book can well be recommended to those interested in military medicine.

Neurology: Lectures for medical students and general practitioners. By Knud H. Krabbe, M.D. 8°, paper, 387 pp., with 4 illustrations. Copenhagen, Denmark: Einar Munksgaard, 1941. 12 Dan. Cr.

Dr. Krabbe, a well-known neurologist from Copenhagen, has long been a lecturer in the hospital and medical school in that city. He is widely known as a distinguished contributor to the literature on neurology. His lecture notes, slightly expanded and carefully translated, are now published in English, a remarkable contribution from Denmark in a time of great uncertainty, because of the German occupation.

The lectures themselves are effectively compiled, and although they report what must be considered only as routine neurologic information, they are written in a pleasant style, with remarkable completeness of material. The book forms an excellent introduction to the subject and should be widely read in this country by medical students and practitioners. There is a flavor, moreover, of continental neurology, not found in American and English textbooks. One should not look, however, for a complete treatise on the subject. These are introductory lectures, but as such are sound, clear and extremely readable. Seldom has the reviewer met with a book by a foreign author that has pleased him more in its English dress. This volume is highly recommended.

In the May 15, 1941, issue of the *Journal*, the price of this book was incorrectly listed as $12.00. It should have been Danish crowns, or about $3.00, which is a reasonable price for an important publication.

Orbital Tumors: Results following the transcranial attack. By Walter E. Dandy, LL.D., M.D. 8°, cloth, 168 pp., with 100 illustrations. New York: Oskar Piest, 1941. $5.00.

Up to comparatively recent times, orbital tumors have been operated on by ophthalmologists, who have used the ordinary orbital approach. With the advent of neurosurgery, it was soon discovered that orbital tumors are often intracranial tumors, with extensions into the orbital space, and thus cannot be entirely removed when operated on through the anterior part of the eye. Nearly 75 per cent of primary orbital tumors are also found in the intracranial chamber. Because of this and also because of the more favorable chance of removing the tumor in the orbital cavity, Dandy advocated the intracranial approach as early as 1921. His point of view has now been widely accepted, and, except for tumors lying in the anterior portion of the right orbit where there is no reason to suspect an intracranial extension, all other tumors of the orbit should be exposed by the neurosurgeon. This, indeed, according to Alan C. Woods, who writes the preface to Dandy's book, has become the routine practice at the Johns Hopkins Hospital. The monograph reports 31 cases, in most of which the tumors were removed by Dandy. The book is finely illustrated, has a good bibliography, and is a splendid example of a small monograph on a single field of neurologic and neurosurgical interest. To neurologists, the book is one of prime importance, and it is hoped that the ophthalmologist, too, will recognize its value.

NOTICES

HARVARD MEDICAL SOCIETY

Beginning October 14, the Harvard Medical Society will meet once a month on the second Tuesday of each

month, with the exception of November, at 8:15 p.m. in the amphitheater of the Peter Bent Brigham Hospital.

The program for the October 14 meeting is as follows:

Clinical presentation.
Recent Advances in Experimental Renal Hypertension. Dr. Lewis Dexter.

JOSEPH H. PRATT DIAGNOSTIC HOSPITAL

Bennet Street, Boston
Lecture Hall, 9–10 a.m.

MEDICAL CONFERENCE PROGRAM, OCTOBER

Wednesday, October 8 — Spleen: Facts and fancies. Dr. William Dameshek.
Friday, October 10 — A Routine Office Cardiovascular Physical Examination. Dr. Samuel A. Levine.
Wednesday, October 15 — A Dental Pediatric Presentation. Dr. F. C. McDonald and Dr. Leonard Despres.
Discussion by Dr. Basil G. Bibby and Dr. Paul Boyle.
Friday, October 17 — Gastric Ulcer. Dr. Sara Jordan.
Wednesday, October 22 — Acne, Hirsutism and Menstrual Disorders. Dr. C. H. Lawrence.
Friday, October 24 — Physical Fitness in Late Maturity. Dr. Robert T. Monroe.
Wednesday, October 29 — X-ray Demonstration. Dr. Alice Ettinger.
Friday, October 31 — The Differential Diagnosis of Hemoglobinuria. Dr. T. Hale Ham.

On Tuesday and Thursday mornings, from 9 to 10 o'clock, Dr. S. J. Thannhauser will give a medical clinic on hospital cases.

On Saturday mornings, from 9 to 10 o'clock, there will be a presentation, with discussion, of dispensary and district cases.

There will be no morning medical conferences the week of November 3, during the meetings of the American College of Surgeons.

JEWISH MEMORIAL HOSPITAL

The first diagnostic therapeutic conference of the Jewish Memorial Hospital will be held at the hospital, 55 Townsend Street, Roxbury, on Thursday, October 9, at 11 a.m. Dr. Reginald Fitz will speak on "Heart Disease."

Interested physicians and medical students are cordially invited to attend.

CONSULTATION CLINICS FOR CRIPPLED CHILDREN

The date of the consultation clinic for crippled children, to be held in Gardner, has been changed from October 14 to October 21. The dates of the other clinics, as announced in the September 25 issue of the *Journal*, remain unchanged.

SUFFOLK DISTRICT MEDICAL SOCIETY

During 1941–1942, the Suffolk District Medical Society will hold two meetings, instead of three as in previous years. Both meetings are business meetings. The executive committee may, for business or scientific reasons, announce additional meetings during the coming year.

Wednesday, October 22, Boston Medical Library. 8:15 p.m. "Personal Experiences in Unoccupied France, 1941." Dr. Harold C. Stuart.

Wednesday, April 29. Annual meeting. Boston Medical Library. 8:15 p.m. Election of officers. "Virus Infections of the Central Nervous System." Drs. Houston Merritt and John H. Dingle.

BOSTON DOCTORS' SYMPHONY ORCHESTRA

The Boston Doctors' Symphony Orchestra will resume rehearsals, under Alexander Thiede, on Thursday, October 9, at 8:30 p.m. at Station WMEX, 70 Brookline Avenue, Boston. Those interested in becoming members should communicate with Dr. Julius Loman, 520 Beacon Street, Boston (KEN 3200 or LON 2155).

AMERICAN SOCIAL HYGIENE ASSOCIATION

The American Social Hygiene Association is arranging a dinner to mark the thirtieth anniversary of the announcement of the discovery of salvarsan by Dr. Paul Ehrlich. Mrs. Ehrlich will be the guest of honor on this occasion. Among the other speakers will be Dr. Thomas Parran, surgeon general of the United States Public Health Service. The Honorable Frances Payne Bolton will preside.

The dinner will be held in the Jade Room of the Waldorf-Astoria, on Saturday evening, October 11, at 7:30. Tickets may be obtained from the American Social Hygiene Association, 1790 Broadway, New York City, at a cost of $3.85, including gratuity.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING SUNDAY, OCTOBER 5

MONDAY, OCTOBER 6

12:15–1:15 p.m. Clinicopathological conference. Peter Bent Brigham Hospital amphitheater.

TUESDAY, OCTOBER 7

*9–10 a.m. Medical clinic. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

12:15–1:15 p.m. Clinicoroentgenological conference. Peter Bent Brigham Hospital amphitheater.

WEDNESDAY, OCTOBER 8

American Academy of Pediatrics. Hotel Statler, Boston.

*9–10 a.m. Spleen: Facts and fancies. Dr. William Dameshek. Joseph H. Pratt Diagnostic Hospital.

*12 m. Clinicopathological conference. Children's Hospital.

2 p.m. New England Dermatological Society. Massachusetts General Hospital, Skin Out-Patient Department.

THURSDAY, OCTOBER 9

*9–10 a.m. Medical clinic. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

American Academy of Pediatrics. Hotel Statler, Boston.

*11 a.m. Heart Disease. Dr. Reginald Fitz. Jewish Memorial Hospital, 55 Townsend Street, Roxbury.

FRIDAY, OCTOBER 10

American Academy of Pediatrics. Hotel Statler, Boston.

*9–10 a.m. A Routine Office Cardiovascular Physical Examination. Dr. Samuel A. Levine. Joseph H. Pratt Diagnostic Hospital.

*8:15 p.m. The Nature and Treatment of Organic Arterial Disease. Dr. Edward Edwards. United States Naval Hospital, Chelsea.

SATURDAY, OCTOBER 11

American Academy of Pediatrics. Hotel Statler, Boston.

*9–10 a.m. Presentation, with discussion, dispensary and district cases. Joseph H. Pratt Diagnostic Hospital.

*Open to the medical profession.

OCTOBER 9–MAY 14. Pentucket Association of Physicians. Page 473, issue of September 18.

OCTOBER 11. American Social Hygiene Association. Page 559.

OCTOBER 13–24. 1941 Graduate Fortnight of the New York Academy of Medicine. Page 834, issue of May 8.

OCTOBER 14. Harvard Medical Society. Page 558.

OCTOBER 14–17. American Public Health Association. Page 579, issue of March 27.

OCTOBER 19–23. American Academy of Ophthalmology and Otolaryngology. Page 350, issue of August 28.

OCTOBER 29–30. New England Postgraduate Assembly. Pages ii-iii, issue of September 11.

OCTOBER 29–NOVEMBER 1. Association of Military Surgeons. Page 473, issue of September 18.

NOVEMBER 3–7. American College of Surgeons. Page vii, issue of July 31.

NOVEMBER 5–6. American Conference on Industrial Health. Page 473, issue of September 18.

JANUARY 3. American Board of Obstetrics and Gynecology. Page 473, issue of September 18.

JANUARY 10–11. Forum on Allergy. Page 392, issue of September 4.

APRIL 20–24. American College of Physicians. Page 996, issue of June 5.

DISTRICT MEDICAL SOCIETIES

BERKSHIRE

OCTOBER 30.
APRIL 30.

BRISTOL NORTH

APRIL 16. Taunton.

ESSEX NORTH

JANUARY 7. Haverhill.
MAY 6. Lawrence.

ESSEX SOUTH

NOVEMBER 12. Beverly Hospital, Beverly.
DECEMBER 3. Salem Hospital, Salem.
JANUARY 7. Danvers State Hospital, Hathorne.
FEBRUARY 11. Lynn Hospital, Lynn.
MARCH 4. Essex Sanatorium, Middleton.
APRIL 1. Addison Gilbert Hospital, Gloucester.
MAY 13. Annual meeting (place to be announced).

FRANKLIN

NOVEMBER 11.
JANUARY 13.
MARCH 10.
MAY 12. Annual meeting.
Meetings will be held at the Franklin County Hospital at 11:00 a.m.

HAMPSHIRE

NOVEMBER 5. Veterans Hospital, Leeds, 4:30 and 6:00 p.m.
JANUARY 7. Belchertown State Hospital, 1:00 and 4:00 p.m.
MARCH 4. Hotel Northampton, Northampton, 4:30 and 6:30 p.m.
MAY 6. Hotel Northampton, Northampton, 8:30 p.m.

MIDDLESEX EAST

NOVEMBER 12.
JANUARY 28.
MARCH 18.
MAY 6.

All meetings will be held at 12:15 p.m. at the Bear Hill Golf Club, Stoneham, except that of May 6, which will be held at Woburn at 6:30 p.m.

MIDDLESEX NORTH

OCTOBER 29.
JANUARY 28.
APRIL 29.

NORFOLK SOUTH

NOVEMBER 6.
DECEMBER 4.
JANUARY 8.
FEBRUARY 5.
MARCH 5.
APRIL 2.
MAY 7.

All meetings will be held at 12:00 noon at the Norfolk County Hospital, South Braintree, with the exception of that of February 5, which will be held at the Quincy City Hospital, Quincy.

PLYMOUTH

OCTOBER 16. Moore Hospital, Brockton.
NOVEMBER 20. Plymouth County Hospital, South Hanson.
JANUARY 15. Brockton Hospital, Brockton.
FEBRUARY 19. Jordan Hospital, Plymouth.
MARCH 19. Goddard Hospital, Brockton.
APRIL 16. Bridgewater State Farm, Bridgewater.
MAY 21. Lakeville Sanatorium, Middleboro.

SUFFOLK

OCTOBER 22. Page 559.
APRIL 29. Annual meeting. 8:15 p.m., Boston Medical Library.

WORCESTER

OCTOBER 8. Rutland State Hospital, Rutland.
NOVEMBER 12. Grafton State Hospital, North Grafton.
DECEMBER 10. Worcester City Hospital, Worcester.
JANUARY 14. St. Vincent Hospital, Worcester.
FEBRUARY 11. Worcester State Hospital, Worcester.
MARCH 11. Memorial Hospital, Worcester.
APRIL 8. Hahnemann Hospital, Worcester.
MAY 13. Annual meeting, Worcester Country Club, Worcester.

WORCESTER NORTH

OCTOBER 22. State Hospital, East Gardner.
JANUARY 28. Leominster Hospital, Leominster.
APRIL 22. Burbank Hospital, Fitchburg.
JULY 22. Henry Heywood Memorial Hospital, Gardner.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

The Care of the Aged (Geriatrics). By Malford W. Thewlis, M.D., attending specialist in general medicine, United States Public Health Hospitals, New York City, attending physician, South County Hospital, Wakefield, Rhode Island, and special consultant, Rhode Island Department of Public Health. Third edition, entirely rewritten. 8°, cloth, 579 pp., with 50 illustrations. St. Louis: The C. V. Mosby Company, 1941. $6.00.

Effective Living. By C. E. Turner, Sc.D., Dr.P.H., professor of biology and public health, Massachusetts Institute of Technology; and Elizabeth McHose, M.A., director of physical education for girls and chairman of the Health Council, Senior High School, Reading, Pennsylvania. 8°, cloth, 432 pp., with 164 illustrations. St. Louis: The C. V. Mosby Company, 1941. $1.90.

Clinical and Experimental Investigations on the Genital Functions and Their Hormonal Regulation. By Bernhard Zondek. 8°, cloth, 264 pp., with 59 illustrations and 44 tables. Baltimore: Williams and Wilkins Company, 1941. $4.50.

Studies from The Rockefeller Institute for Medical Research. Reprints, Vol. 118. 4°, paper, 628 pp., with 141 illustrations, 20 plates, 80 tables and 18 charts. New York: Rockefeller Institute for Medical Research, 1941. $2.00.